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ON

TREATMENT, MEDICINE, SURGERY, NEUROLOGY, PEDIATRICS, OBSTETRICS, GYNECOLOGY, ORTHOPÆDICS, PATHOLOGY, DERMATOLOGY, OPHTHALMOLOGY, OTOLOGY, RHINOLOGY, LARYNGOLOGY, HYGIENE, AND OTHER TOPICS OF INTEREST TO STUDENTS AND PRACTITIONERS

BY LEADING MEMBERS OF THE MEDICAL PROFESSION
THROUGHOUT THE WORLD

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Treatment

THE VACCINE TREATMENT OF INFECTIOUS DISEASES

BY RUFUS I. COLE, M.D.

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Common experience tells us that following an attack of certain of the acute infectious diseases, there remains a certain amount of resistance to a second attack of the same disease. The attempt to apply this experience practically began with the vaccination against smallpox. Here the occurrence of a mild disease, vaccinia, due to an attenuated virus, probably identical with that causing smallpox, or closely related to it, is followed by an immunity to the more severe and dangerous disease. With the discovery of the etiological agents in certain infectious diseases, it was but a step to attempt to induce immunity from other diseases by the artificial inoculation of attenuated or killed cultures of the microörganisms in question. This was undertaken by Pasteur, and a considerable amount of success was obtained, especially in certain diseases of animals, as chicken cholera. The attenuation of the organisms was attempted by various measures, especially by exposure to harmful influences, such as heat, sunlight, chemicals, etc. The production of the mild infection was also attempted by means of non-fatal doses of the living organisms in question, and finally by the inoculation of the killed bacteria. While production of immunity by these various measures reached a considerable degree of success in certain diseases, in others, for reasons which are still obscure or which cannot be entered into here, it proved impracticable. Of late years, however, immunization to cholera in this way has been successfully employed by Haffkine, and to typhoid by Wright and Pfeiffer. Such methods, however, can never receive any extensive application in man, as it would necessitate his inoculation with all kinds of vaccines, and this at fairly frequent intervals, as this form of im-

munity is not very lasting. It is therefore evident that to be of service, such immunization processes would have to be efficacious after infection has already taken place. This was early recognized by Pasteur and he made efforts to induce such immunity in animals and man during the course of the disease, but before it had reached its acme. It is probably by an analogous process of immunization of the body during infectious diseases that recovery naturally occurs, and the attempt to produce artificial immunity in infectious diseases is therefore but an attempt to hasten and strengthen the natural processes of the body. Pasteur had success, however, only in one disease, namely hydrophobia. It is quite evident, however, that this disease has certain peculiarities which render it especially suitable for such a form of treatment. It is a disease having a long period of incubation and one in which the date of infection can usually accurately be determined, and so the immunization be begun very early. This is of importance, for the first experiments showed that a certain amount of time is necessary to bring about the required reaction. Since in most diseases immunization can only be begun after the disease is manifest, it was decided that the inoculation of attenuated cultures would only lead to the superimposing of a mild infection upon the more severe one, and the production of still more harmful effects. For a long time, therefore, this method of treating infectious diseases was given up, especially as experiments on animals gave very unpromising results. It is quite evident, however, that experimental infections in animals, induced by the inoculation of lethal doses of the bacteria, are quite different from the ordinary occurrence in man, where usually the grade of infection is moderate, the mortality is relatively low, and where in most cases the body unaided is able to produce a sufficient degree of immunization. Further work also on the nature and details of the immunity process has given new hope that such efforts at cure may be of value, in spite of the theoretical objections which at first seemed unsurmountable.

During the past twenty-five years there have developed two main views as to the nature of immunity, based on which two schools have arisen. One of these, led by Metchnikoff, has maintained that the main defensive powers of the body are the leucocytes, and that the resistance of the individual depends upon the relative

activity of these in engulfing the invading parasites, and so rendering them harmless. The second school, of which Ehrlich has been a foremost leader, has believed that the leucocytes play only a secondary rôle, and that the body fluids, especially the blood serum, contain substances harmful to the bacteria, substances which are increased during the processes of immunization and cure, and are then able to destroy the infectious agents. Metchnikoff, while not denying the presence of such bactericidal or bacteriolytic substances, nevertheless maintained that the chief rôle in immunity resides in the leucocytes.

A discovery by certain French observers, Denys and LeClef, Bordet and others, that the presence of the blood serum was necessary, or at least advantageous, for the ingestion of bacteria by leucocytes, brought a certain degree of harmony between these views. They thought substances were present in the serum which stimulated the leucocytes to phagocytosis, and hence were called "stimulins." It remained for Wright, however, to show that these substances in the serum which were advantageous to phagocytosis do not act upon the leucocytes, but upon the bacteria, and so change them that they may be taken up by the leucocytes. To these substances Wright gave the name of opsonins (*opsono,* "I cater for"). The experiment by which Wright reached this conclusion has been repeated many times by other observers, and its significance cannot be doubted. The experiment is briefly as follows. Leucocytes are obtained from the circulating blood, and by repeated washing in salt solution they are freed from all serum. If now, to a suspension of such leucocytes in salt solution, bacteria are added, no phagocytosis occurs. If, however, to the suspension of leucocytes an equal quantity of serum be first added, and then the bacteria, phagocytosis goes on actively. But, if the serum be added to the bacteria alone, and left in contact with them for a short time, the bacteria may then be repeatedly washed in salt solution, so as to remove all trace of serum, and still active phagocytosis will go on when such bacteria are added to leucocytes in salt solution, without any serum being present. This seems to prove conclusively that the bacteria have been so changed by the serum that the previously resistant bacteria are now rendered susceptible to phagocytosis. In order to show that this particular property of the serum plays any part

in immunity, it is necessary to show that the amount of the opsonic susbtances in the blood varies from time to time and is influenced by infection, either natural or induced.

This demonstration of the variability of these substances was made possible by the application and development of a method devised by Leishmann. The method is well described in an article by Hollister.[1] In brief it is as follows. Equal quantities of a suspension of leucocytes, serum, and an emulsion of bacteria are mixed together, placed in the incubator for a short time, and then the average number of bacteria taken up by the leucocytes is determined by counting in a stained preparation made from the mixture. At the same time that a preparation is being made with the serum which is being tested, a second preparation is made under exactly the same conditions with a supposedly normal serum. The variation of the first serum is determined by a comparison of the results obtained in counts from the two mixtures.

The leucocytes are best obtained by allowing about 1 c.c. of blood to flow directly into a tube containing a 1.5 per cent. solution of sodium citrate. The latter prevents coagulation, and the blood corpuscles may be thrown to the bottom by centrifugalization. The supernatant fluid is poured off and replaced by normal salt solution. The corpuscles are then mixed in this, and again centrifugalized to free them from all plasma. On centrifugalization it is found that the red cells sink to the bottom, and the leucocytes form a thin layer above them. These can then be obtained, mixed with some red cells, by means of a small capillary pipette. The serum is obtained in small glass bulbs drawn into capillary tubes, in much the same manner that serum is obtained for the Widal test. The bacterial emulsion is made by thoroughly mixing in normal salt solution a small amount of growth on agar of the bacterium to be employed. A piece of glass tubing is then drawn out into a capillary extremity, the end of the capillary tube is cut off squarely, and a mark is made with a glass pencil a short distance, about an inch, from the end. By means of a rubber teat a quantity of the leucocytic emulsion is drawn into the capillary tube exactly to the point marked. A small amount of air is then allowed to enter the tube

[1] Surgery, Gynecology and Obstetrics, 1906, iii, 740.

and the same process is repeated with the bacterial emulsion. Again a small bubble of air is allowed to enter, and lastly the serum is drawn into the tube to the same point. By forcing the contents of the tube into a watch glass and mixing, a mixture containing equal quantities of the leucocytic emulsion, bacterial emulsion and serum is obtained. This mixture is then drawn into the capillary tube, the end of the tube is closed by sealing in the flame and the tube placed in the incubator for 15 minutes. The sealed end of the tube is now broken, the contents forced into a watch glass and again mixed. Smears are then made on slides as is done in making ordinary blood preparations. It will be found by proper methods that most of the leucocytes will be drawn to one end of the slide, and the counting so facilitated. The usual stain employed is some modification of the Romanowski stain, or if tubercle bacilli are used, carbol-fuchsin is employed, with counter-staining by methylene blue. Suppose that within 50 leucocytes, which is the number usually studied, 300 bacteria are counted. This would give an average of 6 bacteria per leucocyte, and this figure is spoken of as the phagocytic index. Now suppose that in the preparation made in exactly the same way, with normal serum instead of the test serum, the phagocytic index is found to be 12. The test serum is, therefore, thought to contain less opsonin than the normal, and the ratio between the two, the abnormal divided by the normal, 6/12 or .5, is spoken of as the opsonic index.

By means of this method Wright has demonstrated that the amount of opsonin in the serum varies from time to time, and has also shown that upon the injection of bacteria killed at 60°, the amount of opsonin in the blood at first falls. Later in response to the stimulation, the organism reacts by increasing the amount of opsonin in the blood, and the index then rises. Wright has inferred that the relative grade of immunity of the body to the infecting organism depends upon the amount of opsonin in the blood. He therefore thinks that by the subcutaneous injection of killed bacteria, the resistance of the body to infection can be increased, and thus the method be made use of in treatment. It must be borne in mind, however, that as we have stated previously, the idea of the treatment of infectious diseases by vaccines antedates the discovery of opsonins. In fact, Wright had previously employed the vaccine

method in the treatment of acne and furunculosis, and the discovery of opsonins was made while attempting to find a method by which he could control the administration of such vaccines. He had tried the estimation of the amount of agglutinin and of bactericidal substances in the blood for this purpose, but found these methods unsuitable. This fact is of importance, making manifest, as it does, that the principle of vaccination is independent of the truth or falsity of the opsonic theory.

This theory, however, has much simplified the conception of the process of immunization, and has theoretically made much more practicable this method of treatment. According to the earlier views, the effort in treatment was directed toward the immunization of man after injection, by means of gradually increasing doses of the bacteria, just as is done in animals in order to render them immune before infection. In the attempt to produce opsonic immunity, however, the procedure is different, for it was early discovered that while the amount of opsonins in the blood apparently varies in different persons, and while the amount differs in the infected from that in the uninfected, these differences are not of very high degree. Occasionally indices as low as .3, or as high as 3 are obtained, but in most cases the variations are between 0.5 and 1.5. Furthermore, the injection of vaccines causes the index to rise, but this elevation is relatively slight and only temporarily sustained.

On the other hand, by the injection of toxin or bacteria into an animal, the antitoxic substances, or agglutinins and bactericidal substances, respectively, may be made to rise in a step-like manner, and by properly interspacing the doses, enormous increase of these substances in the blood may be brought about. Furthermore, it was shown by Ehrlich and Brieger in 1892 that when tetanus toxin is introduced into an animal, there occurs at first a slight diminution of antitoxic substances in the milk, and this is followed by a rise. Solomonson and Madsen later showed that this was true of the blood of animals when diphtheria toxin was introduced. The production of other immune substances, agglutinins, bactericidal substances, etc., follows much the same course.

Wright made practically no observations on the process of opsonic immunization in animals, but from his study of the changes in the opsonins in the serum of man following the inoculation of

dead cultures, he showed that there first occurs a diminution in the amount of opsonins in the blood, which decrease may last for forty-eight hours or longer, a period of so-called negative phase, followed by an elevation of the index, a so-called positive phase.

This positive phase, however, is short, lasting usually from one to two weeks, or less, when the index falls to the previous level. Furthermore, it was found that by a second injection during the positive phase a further slight rise might be obtained, which Wright speaks of as summation of positive phases. On the other hand, Wright showed that by injecting during the negative phase, a second negative phase may be added to the first one and so harm be done. But by no method of interspacing doses has he succeeded in raising the index to any considerable degree. Even by a summation of positive phases, Wright could only bring the index up to 2, 3 or in very rare cases 4. This is manifestly a very great difference from the results obtained in the attempt to produce other immune bodies, where they may be increased thousands or millions of times. Therefore, Wright does not attempt to produce a high degree of immunity during the treatment, as this is impossible, but, by the proper interspacing of doses, attempts to keep the opsonic index above the normal, as high as possible, of course, but without attempting to reach any high grade of opsonic immunity. For this reason, as well as the fact that large doses produce a greater negative phase, very small amounts of the vaccine are employed. A knowledge of the principles which we have enunciated is necessary for the proper carrying out of the process according to the Wright method.

Certain criticisms may be raised to the principles and method as laid down by Wright.

In the first place, before these principles are considered as established, one must be sure that the method of estimating the opsonic index is accurate. We cannot here go into all the objections to the method. It is exceedingly complicated and a great deal of experience is required before even fairly consistent results can be obtained. Various modifications and improvements in the technique have been devised with a view to rendering the results more accurate, but one must admit that even in the most experienced hands, there is still a considerable limit of error. Whether this

limit is sufficient to invalidate the conclusions which have been drawn remains to be seen.

Secondly, it must be stated that the injections of vaccines do not lead to the changes in the opsonic index as described by Wright, with as great regularity as might be desired. Even the curves obtained by Wright show a bewildering irregularity. It has been shown and accepted by Wright that many non-specific processes may materially modify the opsonic index. Menstruation, exercise, even the taking of food, have been found, by various observers, to modify the opsonic index to as great an extent as does infection or vaccination.

Admitting, also, that vaccination and infection cause a modification of the amount of opsonins in the blood, it does not necessarily follow that these substances play any considerable part in immunity. Many of the objections that have been raised to the phagocytic theory may be raised to the opsonic theory, for the latter is but a modification of the former. The idea that the leucocytes play any important rôle in the immunity process is contrary to our present views of the infectious process in diseases like tuberculosis and typhoid. As regards the question of treatment by vaccines, however, the decision as to whether the method is of value or not must, as Wright states, rest upon clinical results. Should vaccinations prove valuable this will not necessarily prove that the opsonic theory is true, until it is shown that just as good results are not obtained by giving the vaccines without reference to the variations in opsonic content. Clinical experience, however, is fallacious, and critical judgment must be brought to bear upon the results. One needs only to review the German literature of 1891, following the publication of Koch on tuberculin, to see how even medical men may at first be deceived as to the value of a remedy.

We believe, however, that sufficient clinical evidence has been presented to make it more than justifiable to employ this method, but we should guard against too hasty conclusions. Above all it should be insisted upon that the opsonic index is of absolutely no value unless carried out with the greatest care and by one having had considerable experience. That good clinical results have been reported under conditions where the accurate determination of the index was not likely, makes it probable that either one of two things

is true; either conclusions have been drawn from too few cases, or the opsonic index is not of great importance for the proper interspacing of doses.

As to the best procedure in carrying out the treament by this method, we may say that the method is too new and modifications have arisen with too much rapidity to make it possible to lay down any definite rules. As the method has been championed by Wright, however, it seems no more than fair that those who employ it, especially with a view of convincing themselves of its accuracy and importance, should follow his directions.

Vaccines have been employed most largely in the treatment of chronic staphylococcus infections, such as acne and furunculosis, gonococcus infections, to a less extent in streptoccocus infections, in colon bacillus infection, tuberculosis, and in isolated instances in almost all infections in which the etiological agent has been isolated. The vaccines for coccus infections, colon bacillus infections, etc., are made by growing the bacteria on agar, making an emulsion in salt solution, and then heating in a closed tube to 65° for one hour. The number of bacteria contained in 1 cc. of the vaccine is estimated by means of a method devised by Wright.

The dose is estimated in numbers of bacteria. The dose for staphylococci is usually 500 to 2500 million, streptococci 200 to 1000 million, gonococci 200 to 1000 million, B. coli, 100 to 1000 million. In the treatment of tuberculosis, Koch's new tuberculin TR is employed in doses of 1/1000 to 1/400 mgr. We have previously stated the principles which guide one in the proper interspacing of doses.

It is at present difficult to judge of the results of this treatment from a study of the published reports. The writers have usually reported isolated cases, and it is not yet possible to compile statistics, nor are we yet able to draw any accurate conclusions from our own cases treated at the Johns Hopkins Hospital. The construction of opsonic index charts is long and tedious, and where this method is strictly adhered to in the treatment of cases, a single individual is able to perform the work necessary, only in a few cases, during a single period of time.

Staphylococcus Infections.—In certain cases of acne we have seen apparent benefit, yet the results have not been absolutely con-

vincing. A review of the literature shows that most of the cases are reported as having been improved by the treatment, and this in general has been our experience, complete recoveries, however, being rare. Certain of the patients with acne have thought themselves much improved at times, but where followed long enough, and where all other forms of treatment have been omitted, there has usually been a recurrence of the original trouble in spite of vaccination. Certain cases of furunculosis have recovered, but one must bear in mind that most cases of furunculosis tend to recover spontaneously with no treatment whatever. And, on the other hand, I know of one case at least, in which the treatment has been carried out consistently for five months with boils still recurring.

Gonococcus Infections.—Dr. Meakins and the writer have, during the past five months, treated about twenty cases of gonorrhœal arthritis, fifteen of them being carried over a long enough period of time to judge of results. These cases are all published in considerable detail in the *Johns Hopkins Hospital Bulletin* for June, 1907. Results have been encouraging, both in the more acute and also in the very chronic cases. All the cases recovered without permanent damage to the joints, and in certain of the cases where there was stiffness present before treatment, marked improvement has occurred. Nevertheless, the number of cases is still too few for us to decide definitely as to how much of the good effects are to be attributed to the vaccine. It is our decided impression that the method has been of value and that the results obtained have been much better than we have seen in similar cases previously treated by other methods. In these cases we have followed quite accurately the course of the opsonic index, and have interspaced our doses in accordance with the findings; as a general rule, however, we have given the vaccine about every ten days, and we now feel that possibly as good results might have been obtained without paying any attention to the opsonic index. If we felt great confidence in the accuracy of the index as obtained by the present method, we should feel justified in continuing, notwithstanding the great labor involved. For those who are studying the matter, especially with a view of reaching definite conclusions we would advise continuing the estimation of the indices and regulation of the dosage by them; but where the vaccines are given in a very tentative

manner, we should think it justifiable to administer them at intervals of about ten days, depending upon the clinical results.

Streptococcus Infections.—Wright and others have reported the successful treatment of streptococcus septicæmia by means of streptococcus vaccine. The theoretical objection might be raised to such treatment, that by the administration of dead cocci, we are simply adding to the sum total of the infection, since where bacteria are circulating in the blood, we cannot hope to cause a greater stimulation to the protective forces than is already present. There is some evidence, however, to justify this course, since, as is well known, the administration of diphtheria toxin subcutaneously gives rise to a much more extensive formation of antitoxin than does the inoculation of the toxin directly into the circulation. In the production of hæmolysins also, experience has shown that subcutaneous injection is in most cases preferable to intravenous. Whether, however, from the cases so far reported we can judge as to the value of the vaccine, seems doubtful. It must be remembered that many cases of streptococcus infection, even where the bacteria are cultivated from the circulating blood, recover, where no specific treatment whatever is employed. We have had one case of streptococcus septicæmia with arthritis, pericarditis and later pneumonia, which we treated with vaccines and which recovered, notwithstanding the fact that she was desperately ill when the treatment was commenced. On the other hand, in a second case, from which we obtained blood cultures, we had prepared vaccine and were about to make our first injection, when on going to the hospital for that purpose, we found the patient's temperature was lower and she made an uninterrupted recovery without any specific vaccination treatment whatever.

Tuberculosis.—So far not much has been done in the treatment of pulmonary tuberculosis, and, considering the marked rôle which it is now recognized that secondary invaders play in the course and symptoms of this disease, it is apparent that theoretically such attempts at treatment would not be very efficacious. Tuberculosis, however, localized in the interior of the body, such as in the glands and joints and bones, would seem to offer ideal conditions for the application of this method. Here we have a latent chronic infection, not giving rise to much reaction on the part of the body as a whole,

and it would therefore seem that by the injection of stimulating substances, we might increase the immunity of the body sufficiently to overcome the infection. In six cases of joint tuberculosis which we have treated by this method, the results have been very good in all, so far as the symptoms and apparent course of the disease are concerned. In none of the cases, however, so far, is the disease absolutely cured, although some of the patients have been under treatment for four months. The same is true of tuberculous glands of the neck. Two of our cases have been of great interest as showing the probable effect of fresh air and hygenic measures when combined with the vaccine treatment. These two patients suffered from enlarged glands of the neck in about the same stage, and treatment was commenced at about the same time. One of the patients has been able to remain out of doors almost constantly, sleeping out at night, has been quiet and has received good food. There has been very marked improvement and the enlargement of the glands is rapidly disappearing. The other patient was an operator in a telephone office, would not sleep out of doors at night, had insufficient food, and in her case the glands have continued to enlarge, and it has been decided advisable to remove them surgically. The opsonic curve in these two cases shows no material differences, and the reaction to the vaccine, as indicated by the index, was fairly good in both cases.

It is impossible to enter here into all the variety of cases in which vaccine treatment has been thought advisable and has been carried out. If one may judge from the reports, the results have been excellent in many conditions.

As to the conclusions which we may reach from the study of the subject so far, we may say that it is demonstrated beyond a doubt that the presence of serum is advantageous and in most cases essential for phagocytosis; that the substances of the serum which are necessary for this process act upon the bacteria; and, lastly, that variations in the amount of these substances in the serum occur from time to time and that they may probably be somewhat increased by the injection of a suitable vaccine.

As regards the value of treatment by means of vaccines, this is manifestly an entirely different matter from the question of opsonic immunization. We may say that clinical results indicate

that the method is of value. Since, in the present movement, during which time very good results seem to have been obtained, small doses have been used, it seems probable that in the use of these small doses a decided advance has been made in the treatment of acute infectious diseases and especially in the method of using tuberculin. As to whether our efforts in the treatment of acute infectious diseases by vaccines should be made with reference to the increase of opsonins in the blood, or as to whether we have at present, methods sufficiently accurate for determining the exact amount of these substances, future experience must show; but we must remember that even though the method for determining opsonic indices should prove inaccurate, and though opsonins themselves may be shown to have no important part in immunity, vaccination in the treatment of infectious diseases will not necessarily be completely invalidated thereby. The results are hopeful and the method should be continued until we have absolute data as to its value. We must constantly bear in mind, however, the principles on which the method is based. It is also important to remember that whatever increases our knowledge of the process of immunizaton will aid us in the solution of that most important problem in medicine—the treatment of infectious diseases.

THE TREATMENT OF RHEUMATISM *

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Our subject this evening is a very practical one, and I do not propose to detain you with many theoretical considerations; it is, however, first necessary to say a word with regard to the causes or suggested causes of rheumatism.

As you know, it has now been believed for many years that rheumatism is an infectious disease, and I think we all agree with regard to this. But as regards the rival organisms which are claimed as the actual cause of this condition there is very little agreement, and there are many who believe that no one has, so far, substantiated his claim. Here in England we have been hearing a good deal about a particular diplococcus or streptococcus, which was originally discovered many years ago by Triboulet in France. It was described later by Wassermann and others in Germany. I am not at all clear that this organism has anything to do with rheumatism. You can find it in a certain number of cases of malignant or infective types of cardiac disease. I have such a case at present in the wards from which this organism has been recovered, but I do not think for one moment that it is the cause of rheumatism. Eminent authorities on bacteriology have tried to obtain this diplococcus from cases of uncomplicated rheumatism, with invariable failure; and, moreover, measures based upon a belief in it have proved of no value in the treatment of rheumatism. It is a micro-organism which does not satisfy Koch's laws, and which has not led to improvement in treatment. It is surely unscientific to argue that because a microbe is found in cases of a disease which is complicated by superadded infective processes, it is therefore to be regarded as the cause of the disease itself.

* Lecture given at the Polyclinic on January 24, 1906.

It is customary to consider rheumatism separately as it affects children and adults, and I do not wish to depart from this habit, although I think in some cases this division may be shown to have done much more harm than good.

Rheumatism in Children.—We must remember first in connection with children, that instead of having an acute attack of joint rheumatism, they are liable to the incidence of various conditions belonging to what is known as the rheumatic cycle. A child may have an attack of tonsillitis, frequently repeated, and having failed in health, may be found to have endocarditis. Or he may suffer from "growing pains," and suddenly develop chorea. Or, without any preliminary warning whatsoever, the little patient may be found to be suffering from endocarditis or its effects. There is a series, a cycle of changes, any one of which may appear first, and be followed by some or all of the others in any order. As you all know, rheumatism in children is not so markedly a joint affection as in adults. It is an affection of the serous membranes in general, rather than of the joints in particular.

In connection with this I ought to say a word about the relation of tonsillitis to rheumatism in children, which is a subject of the greatest importance. There are at least three ways in which tonsillitis may be related to rheumatism: (1) An attack of tonsillitis, either lacunar or parenchymatous (quinsy), may definitely precede an attack of rheumatism by any length of time from six or seven days to six weeks, ten days being the common interval; (2) at the beginning of an attack of rheumatism there may be a sore throat, slight in degree, a uniform redness, or—as I prefer to call it—a faucial erythema; (3) the subjects of rheumatism may suffer from chronic hypertrophy of the tonsils. With regard to the relation of these to rheumatism, I think we may at once dismiss the last two. The faucial erythema is merely an early symptom of rheumatism. This rash of the throat appears in the same way as the other erythemata of the early stages of attacks of rheumatism. Chronic hypertrophy of the tonsils, on the other hand, is a result of repeated attacks of more or less acute tonsillitis, and bears no definite relation to the breaking out of joint symptoms. The lacunar or parenchymatous tonsillitis, however, which precedes the attack, is of the greatest possible importance. It is at present fashionable to say that

the tonsils are frequently the seats of infection in many diseases. I beg leave to differ from that view in most cases. I do not believe, for instance, that the fact that giant cells are found in the tonsils is any evidence whatsoever that the body is infected with tuberculosis through the tonsils. It is here evident that the tonsils have been attempting to defend the body against the onset of micro-organisms which have entered through cracks in the mucous membrane or other places. It would be just as reasonable to condemn the tonsils on this evidence as the cause of tuberculosis as it would be to imprison a policeman because you find a jemmy in his hand which he has removed from a burglar. Nor am I personally prepared to condemn the tonsils in the case of rheumatism which has been preceded by tonsillitis. I believe that in a certain number of cases, in which the tonsils have been repeatedly inflamed, they may have lost the power to withstand the entrance of the rheumatic virus, but I do not think we have any right to say that the tonsils are the site of the infection in rheumatism. If that were so, it would be an argument for the removal of the tonsils in all normal children. It is quite possible, on the other hand, that the tonsils may attempt to defend the body from the effects of oral infection by the rheumatic virus.

The increase of our knowledge with reference to the symptomatology of rheumatism in children has been accompanied with great advantages, and also with great disadvantages; the latter have become particularly obvious in the matter of treatment. Twenty years ago the existence of rheumatism in children was hardly recognized, while now it has been definitely established that there is great incidence of rheumatism in childhood, and that it is one of the most common causes of cardiac disease. In another way, this increase of knowledge is extremely dangerous, because now, directly a child has anything which may be assumed to be rheumatism, there is only too great a liability to dose it with salicylates, and to do nothing more. Salicylates relieve rheumatic pains, lower temperature, and ease the joints; but they are entirely without action on the most dangerous complication of rheumatism, cardiac inflammation. There is no doubt that a child with "growing pains," who is at once treated with salicylates, will recover from the growing pains, but I believe that to treat a child in this way *only*, is to do more harm than good. It may be advisable to give salicylates for the time being,

but the fact that a child has had rheumatism indicates a totally different line of treatment in what I may call the interparoxysmal stage. Every child who has had an attack of rheumatism is a suspect, is liable to explode with an attack of endocarditis, and only to emerge from it with a permanently crippled heart; and I most strongly maintain that such children should be looked after from the time of the incidence of an attack of rheumatism to the time when the process of growth is completed. Not very long ago a medical friend consulted me about a son who had had several attacks of sore throat, had had one attack of rheumatism, and had a slight mitral murmur. The boy was just at an age to go to school, and I suggested that it would be a good thing if he did not do so. It so happened that my advice was disregarded, owing mainly to a chain of circumstances with which I need not trouble you now. The boy went to a public school, had a fresh attack of endocarditis, and died within a year. I believe that a child who has been attacked with rheumatism and suffers from heart complications in consequence, in any case, however well he may appear, ought to be strictly looked after, and regarded as an exotic. He ought not to be overworked mentally or physically: he must be kept under rational conditions, with private teaching if possible, with moderate exercise, and should be in every way regarded as delicate. I cannot too strongly insist upon this. I believe that any number of cases, which emerge from childhood with a permanently crippled heart, would be prevented from sustaining such damage if this very grandmotherly treatment were adopted, and I believe it to be most essential that it should be insisted upon throughout. When a boy has finished growing, and reached the age of twenty, twenty-one, or twenty-two, he can be allowed to develop both mentally and physically, but up to that time make an exotic of him, let him lie fallow, and keep him from any further risk of damage to the heart, and so far as possible from any recrudescence of rheumatic conditions. Unless we apply what we have learnt with regard to rheumatism in children in this way, we shall do more harm than good, by allowing very dangerous complications, which are not amenable to salicylates, to develop unchecked.

Let us consider the treatment of another complication which is common in connection with rheumatism, namely, chorea. This is an intoxication with the rheumatic poison, as a rule acting upon

nerves hereditarily predisposed. The nervous system in these cases is less well-balanced, especially as regards the association of its motor elements, than usual, and more prone to suffer from the action of a virus. I regard it as a strong point in favor of this view that an attack of chorea can undoubtedly be determined by fright. Sir William Gowers records a case of a small boy who was climbing an apple-tree—in pursuit of knowledge—when a farmer appeared with a heavy stick. The boy began to tremble, and the tremor went on to chorea. There is no doubt that, after excluding a vast number of unfounded allegations, a large proportion of cases are determined by fright, fright by fire or by a dog being two of the commonest causes.

You may ask how it is that a definite disease of toxic origin can be brought on by fright. I think one can explain this quite simply by recalling an analogy. You know perfectly well that a chronic alcoholic who has been soaked in the poison for many years, may show no obvious signs of alcoholism. One day he breaks his leg, and the next day he has delirium tremens: or the same phenomenon may follow if he has a chill, and develops pneumonia. Or a diabetic, who has been soaked in a different kind of poison for months or years, takes a fatiguing railway journey, and promptly develops coma. These sudden stimuli are on the same plane, and act in the same way as fright in chorea. The child is, so to speak, supersaturated with the poison, and the addition of the fright or other stimulus causes the chorea, in the same way as a crystal of salt causes a whole mass of magnesium sulphate solution to crystallize. Thus, I think, we have a rational explanation of the determination of these nerve conditions by fright, or other physical or psychical stimuli, although the essential lesions are intoxications.

That being so, how are we to treat chorea? I am afraid I must admit that no amount of reasoning will aid in this matter, and that our knowledge has not borne us beyond the empiricism that we have already followed for many years. I have tried during the last eight years all the remedies which are recommended for chorea, and I find, practically speaking, that one must come back in the end to arsenic, and, moreover, that one reverts to the small dose treatment by arsenic: not treatment with large doses of the drug, but persistent treatment with small doses, associated, of course, with proper rest,

proper nutriment, and with the removal of causes of psychical excitement. I begin with 3 minims thrice daily in a little chloroform water before meals, and gradually increase the dose, but never above 7 minims. I have not yet made up my mind as to whether the strain of school life has an important bearing upon the development of chorea. I am at present engaged in investigating all the cases of chorea in the County Council Schools of London, and when I have finished working them out I shall, perhaps, be in a position to discuss the question. I do not think, as far as I can tell at present, that we can definitely attribute conditions of chorea to overwork at school. I have seen other conditions, such as exophthalmic goître, caused in this way in young women who were preparing for examinations. I do think, however, that children suffering from chorea should be excluded from school, both for their own sakes and for that of the children who may imitate them.

In a severe case of chorea there is no doubt that other remedies may be used, particularly with the object of ensuring rest and sleep. It is even advisable in some cases to keep the patient under chloroform for quite a long time. One sees often in children cases of chorea complicated by definite tremor, and in these cases I have sometimes found that bromides have relieved the tremor, but I have never seen good result from their use in chorea itself.

Acute Rheumatism in Adults.—As regards rheumatism in adults, it is usually divided into acute, subacute, and chronic. You are all familiar with the treatment of acute rheumatism. This leads us to the question of how you should give salicylates: I do not believe that anybody nowadays would think of using any other treatment. As I pointed out a long time ago, a rheumatic sore throat does not yield nearly so well to salicylates as one would expect. Among forms of sore throat which do yield rapidly to this method of treatment is the influenza sore throat, which clears up very quickly under the influence of salicylates. Rheumatic tonsilitis is best dealt with by the use of perchloride of iron and chlorate of potash:

Tinct. ferri perchlor.	10 minims
Pot. chlor.	5 grains
Glycerini	1 drachm
Aq.	ad 1 ounce

M. S.—1 ounce every four hours. To be gargled first and then swallowed.

In treating an attack of acute rheumatism, I personally use ordinary sodium salicylate. I do not think that it has any very great disadvantages in a normal subject, but if given to a person with chronic kidney disease or a failing heart, I have seen unpleasant results. I should not think of using it in such cases.

I do not think that aspirin acts nearly so well in acute joint rheumatism. I find that other more or less insoluble substances, such as salol, are very slow in their action, and not very effectual. Nor am I accustomed to seeing bad effects, such as toxic symptoms, from the administration of sodium salicylate in the way in which I use it. My method is to give 20 grains of salicylate every two hours, with 3 to 5 grains of sodium bromide, made up with quassia. The latter covers the taste very much better than ginger or cinnamon or orange, or any other flavoring; and if one gives the 3 to 5 grains of sodium bromide, I think that one will never see toxic symptoms. I administer this every two hours, and if the patient's temperature and pain have not disappeared in twenty-four hours, I begin to think I have made a mistake in diagnosis, and that something else is present which I had not suspected.

I am quite certain that an attack of uncomplicated acute rheumatism should give way in that time. There is, however, a danger that the patient will, so to speak, get well too soon. If one gives a strong man, who has been very healthy, salicylates in an attack of acute rheumatism, he feels perfectly well in two or three days, and wants to get up and go back to his work. That man is in a dangerous condition, and is exposing himself to the risk of grave complications. This has caused it to be said by some people that the proportion of cardiac complications is greater since salicylates have been employed in treatment than it was before. I keep my patients in bed for ten days at least after the temperature has fallen, reducing the dose of salicylates quite gradually. They do not have meat at this time. Of course, at the end of the ten days or a fortnight they are fed up, and put upon quinine and iron. I believe that if you use salicylates in this way, you will meet with fewer instead of more complications.

There is one complication which we rarely see nowadays, but which was common in my younger days—that is, hyperpyrexia. I believe its disappearance is due to the habitual use of salicylates.

There has been considerable discussion as to local treatment during the attack. As a matter of fact, I do not think you need do anything more than wrap up the joints. There are two schools of thought which advocate further measures in reference to this: on the one hand the use of ice-bags is recommended, and on the other that of blisters. It has been suggested by Dr. Caton that threatening endocarditis may be aborted by the application of fly-blisters to the precordia, but is is very difficult to prove this. It is only by taking a very large proportion of cases, and using them for the purpose of comparison, that one can estimate what proportion will, in the ordinary way, be complicated by endocarditis. I think, however, that the blisters are very good indeed as remedies, in fact one of the best methods of treatment of acute cardiac complications. In pericarditis I hardly ever fail to apply a blister or leeches; I have on several occasions successfully used the actual cautery. With regard to ice-bags my feeling is quite otherwise. I find that they are depressing, uncomfortable, and often ineffectual. I do not think I ever use them now in any inflammatory condition whatsoever, unless it be in cases of meningitis, where one does not employ them so much to relieve inflammation as to mitigate the cerebral symptoms. If an acute cardiac (especially exocardial) complicattion develops, regular local treatment should be employed; in my opinion blisters or leeches, and not cold applications.

The complications that one sees nowadays in acute rheumatism do not usually call for much treatment, but should hyperpyrexia develop, naturally a cold bath is indicated. Whether or not erythema nodosum is a true complication I am not quite sure, but I am not at all confident that it is anything more than a casual epiphenomenon of rheumatism.

Subacute Rheumatism.—Now we come to a more difficult subject, that of subacute rheumatism. This is an expression which we frequently hear, but which is used perhaps more often to cover a multitude of sins, chiefly on the part of the doctor, than for any other purpose. Some years ago I was asked to write an article on muscular rheumatism. I thought I would investigate all the cases of myalgia that I had ever seen, and of which I had notes, and I found that more than three-fifths of them were gouty. Rheumatism does not, as a rule, attack the muscles as often as the fasciæ.

Definite attacks of subacute rheumatism accompanied by very little temperature and slight local symptoms do, however, occur in rheumatic subjects. The joints are not invariably affected in these cases. If they are involved, the interphalangeal joints of the fingers are most frequently attacked. These cases are often seen with swelling of the first and second fingers and especially of the first interphalangeal joints. The joints are very red and tender, and they usually yield at once to a special form of antirheumatic, *i.e.*, salicylate, treatment. Apply an ointment of 1 drachm of methyl salicylate to 1 ounce of lanoline to the joints, cover them with flannel and then with some waterproof tissue, and the fingers will get better in a day or two. This is, as I have said, the commonest situation in which the joints are affected in subacute rheumatism, but the wrists and ankles also are not infrequently attacked. Next to the joints, I should say that the incidence of the disease is most common upon the tendon sheaths, and particularly the tendon sheaths on the dorsum of the foot. Very often one sees people who have had rheumatism, and come back saying that their rheumatism is all right, but that they have something wrong with the instep. The tendon sheaths of the dorsum of the foot have become inflamed. There is no general fever, but general malaise, perhaps, and a little anæmia. The fasciæ also are often affected, and of these one sees the fasciæ of the neck involved most frequently. A sore throat may go on to torticollis, inflammation spreading from the tonsils into the fasciæ between the muscular planes of the neck. An attack of acute torticollis is not muscular but fascial inflammation, and the patient contracts the muscles in order to rest the fasciæ. The sternomastoids in this form of torticollis are not contracted because they are inflamed, but to act as a splint to the inflamed fasciæ.

One is often asked how to diagnose these cases as rheumatism, in distinction from gonorrhœal inflammation of the fasciæ. A rheumatic family history, and a rheumatic personal history, are invaluable, but certain definite sites are affected more often in one case than in the other. In gonorrhœal inflammation the plantar fasciæ are frequently attacked, and there are very often pains in the heel, whereas in rheumatism the fasciæ of the neck are, I think, most frequently involved. Sometimes the erector spinæ muscles may be

attacked in gonorrhœa, but this is infrequent in rheumatism. Inflammation of the sternoclavicular articulation is always gonorrhœal. It may be difficult to diagnose rheumatism from myalgia due to gout, but the history will help, and in the last resort there is always the indication afforded by the response to treatment.

Another form of subacute rheumatism which I do not think is often mentioned, but is increasingly common, is episcleritis, a painful inflammation of the sclerotic, which appears in one quadrant of the eye, spreads over the whole sclerotic, and after three or four days of intense pain, passes away; after a varying interval it appears in the other eye, and so goes on in alternate attacks for perhaps years, unless properly treated. In these cases the condition is quite obvious. The white of the eye becomes dull red in color, and the inflammation spreads from one quadrant to the other. There is usually some tenderness in the supra-orbital nerve, and the affection is almost always brought about in a predisposed subject by slight excess in reading, or it may not infrequently be determined by a little overindulgence in wine. These cases require a different method of administrating salicylates. The fascial rheumatism, rheumatism of the joints, and rheumatism of the tendon sheaths, all require some form of local treatment. For the episcleritis by far the best treatment is aspirin. I am not a great believer in aspirin for rheumatism generally, but in these forms of rheumatism it is most valuable. It is very sparingly soluble: it can be given as a rule in a dose of 10 grains three times a day. The only disadvantage I have seen about it is that it sometimes causes profuse sweating. This appears to be a personal idiosyncrasy, so that I cannot regard aspirin as a definite diaphoretic in all cases. During the attacks, the patient should be put on aspirin, and aspirin should be pushed, if necessary, beginning with 10 grains, and increasing the dose until the pain and the symptoms have disappeared. But in the intervals between the attacks one must also do something, and the proper drug to give almost continuously is salicylate of quinine. Salicylate of quinine is really an insoluble drug, and contains only a small proportion of salicylate. The ordinary compound contains about 2 grains of salicylic acid in every 5, so that one is giving about 3 grains of quinine and 2 grains of salicylic acid. If one administers this in doses of about 6 grains per diem continuously for a

period extending over months or years, one will find that the tendency to subacute attacks gradually diminishes, particularly in the case of episcleritis. People who have for three or four years been crippled by recurrent attacks of subacute rheumatism, have thus been freed from it, and if a slight attack has developed, they have been put upon aspirin, which has cleared it away at once. If one really has a genuine case of subacute rheumatism to deal with, one can usually get rid of the rheumatism entirely by combined treatment with salicylate of quinine and aspirin, and keep it at bay without difficulty so long as the patient will continue the treatment.

Chronic Rheumatism.—Lastly, I have a word to say about chronic rheumatism. Of all the diseases which are useful as cloaks to ignorance, chronic rheumatism is perhaps the most unsatisfactory. We are bound to confess that chronic rheumatism is a disease we do not meet with in practice, a disease which is virtually non-existent. Now and then we see cases of rheumatism in which joints are left permanently crippled. You may remember that I have shown such cases at clinics here, and have pointed out that the condition of the hands and feet is hardly to be distinguished from the juvenile form of rheumatoid arthritis. If you exclude from the category of chronic rheumatism cases of ostearthritis, infective thrombosis, etc., you will find but a very small residuum in which these joint troubles are consequent upon acute rheumatism. There are a few, but they are so rare as to be almost pathological curiosities. The treatment of these very rare cases may be of interest, but is almost devoid of practical importance. It is worth while to mention that the treatment includes hygienic measures, hot air in some form or other, and local massage, and it is often very useful to produce and maintain local anæmia away from the joint, with passive hyperæmia at the joint. This treatment was introduced by Dr. Bier, and is of the greatest value in these cases of true chronic rheumatism.

Such are the general phenomena of a multiform disease, considered as indications for its treatment, and I may repeat my belief that if prophylaxis were in every case properly, systematically, and rigorously carried out, and each development of the disease met by appropriate remedies, we should be a long way on the road towards stamping out, not only rheumatism itself, but also its cardiac complications.

THE THERMAL TREATMENT OF AIX-LES-BAINS

BY JEAN DARDEL, M.D.

Aix-les-Bains, France

The treatment at Aix-les-Bains is generally known, but not so the exact method of applying it. The treatment is chiefly external; the water taken as a beverage is but a secondary factor. The principal and all-important modes of treatment are the bath and the *étuve,* or heated-room; but, above all, the douches, or, as they are properly called (because peculiar to Aix) "Les Douches d'Aix-les-Bains." It is these douches which constitute the treatment of Aix.

The chief object of this paper is to describe precisely the treatment of Aix-les-Bains, and to express succinctly and briefly the mode of action of the waters. To the technic of the Thermal Establishment is, of course, linked the treatment applied outside the Thermal Establishment, which is an integral part of the Cure of Aix-les-Bains.

The *waters* of Aix flow from two springs or sources, which emerge from the Neocomien strata, and which are about one hundred yards from each other. The two sources, the Alum Source and the Sulphur Source, have almost the same chemical composition. It is now admitted that the two sources have a common origin, the waters being conducted by different channels from one and the same reservoir, which reservoir is unique and deep down in the Neocomien strata. The waters of Aix have been put in the sulphurous calciferous class of waters. The water of Aix is a mineral sulphurous water, the amount of mineral matter contained being only 0.49 per litre. It marks 4 degrees in the sufhydrometre Dupasquier. Its density is 1.0024. Besides the mineral elements, the water of Aix contains an organic substance; viz., "la barégine" or "glairine," which gives to the water that unctuous character which the bather so soon perceives. It is the glairine that one sees floating in thin films upon the water of the "piscines," where it is held in suspension in the water. The two sources yield

4,000,000 litres every twenty-four hours. That enormous quantity of water permits a copious use, and the mass employed for each and every operation is one of the important factors of the cure. The temperature of the water is 45 degrees Centigrade. An abundant source of cold water at 11 degrees serves to graduate at will the temperature of the "douche" and the baths.

The *douche* of Aix is a combination of the "douche" and "massage," sometimes called "douche-massage." This douche-massage constitutes the real specialty of Aix-les-Bains. This is not a douche according to the usual signification of the word "douche," for the term implies a colder and a more rapid operation. Nor is it simply a massage. No, it is a special operation performed by a "petrissage," or kneading of the muscles, the patient receiving at the same time a general and abundant douche of the thermal sulphurous water.

The douche is given in rectangular chambers, each chamber having a dressing-room. Some of these chambers are completely closed, while others are open towards the ceiling. This disposition allows the escape of the vapor, and at the same time avoids the over-heating of the atmosphere in the room where the douche is taken.

In each cabin where the douche is administered, the temperature of the water varies from 14 degrees to 43 degrees, and one can have a cold, a warm, or a tepid douche, according to his need or pleasure. But the division called the central division does not admit of any but the thermal water douche.

The conduits which bring the waters, either mineral or cold, run into vessels fixed in the wall in which the waters are mixed. One of these vessels is a reservoir placed at 1.65 metres above ground. The mixing of the waters takes place in the open air, so that the pressure does not vary. This mixed water is destined to flow over the back and the upper parts of the body.

The second vessel for mixing the waters is a closed box, or copper cistern into which the hot and cold waters flow. This mixed water serves to flow over the different limbs of the invalid. In this second reservoir for mixing the waters, the water preserves its full pressure, which varies according to the different stories of the building (14 metres, 9 metres, 6 metres).

The invalid is seated upon a wooden chair, about 1 foot high, furnished with one arm about two-thirds of a foot high, upon which he leans; he rests his feet upon a wooden stool. One of the "doucheurs" places himself before the patient, holding under his arm one of the tubes,—the one which contains both the hot and the cold water. He directs the water over the different parts of the limbs of his patient, at the same time frictioning and petrissing the muscles; and, according to the case, moving the joints about. The second doucheur keeps behind the invalid, holding the tube which contains the water from the free-air vessel. By imperceptible movements he waters the shoulders and the back of the invalid, massaging at the same time the muscles of the neck, back, or trunk. During the whole time of this massaging of the different parts of the body the invalid is inundated, so to speak, with thermal waters.

After a time, which varies according to medical advice, the patient leaves his chair and stretches at full length on an inclined table for massaging. The doucheur, still holding his tube of mineral water, places himself behind the invalid, and proceeds to the massaging of the muscular masses of the back, and of the back parts of the thighs and legs. Massage of the back can also be performed by placing the invalid upon his chair in an inclined position, his hands leaning on the arm of the chair. Certain invalids, who cannot move without great discomfort, are brought to the douche upon a camp-bed, which they are not obliged to leave during the douche, and thus avoid every painful movement.

The massage being terminated, the patient places himself in a corner of the cabin, and there receives the douche in full, or in a shower, or in a sprinkle, which is the most important of all. The last part is extremely important, and the doctors know how to obtain different results, from the use of a broken jet or from a full jet, the jet falling on the patient like a spout, throwing out in full force the water upon him, or coming gently upon him like a shower of rain, either in cold or in warm showers or in Scotch shower-baths or douches.

The douche-massage being completed, the patient is dried, wrapped up in a flannel dressing-gown, and then covered with a woollen rug, put into a sedan-chair, and carried to his hotel. He is

put into bed thus wrapped up, and after twenty minutes or so of heavy perspiration, he is uncovered and again dried, and wiped by the man whose duty it is to help him to get out of the *maillot,* or wraps. This done, the douche-massage is finished.

Occasionally the patient dresses himself at the end of his douche and goes out on foot, to induce perspiration either by exercise or by returning to his bed. But it is preferable to return in the *maillot,* well wrapped up, in the sedan-chair. Formerly this method was used exclusively. Such is the most usual form of the douche of Aix-les-Bains, but its administration may be subjected to certain variations.

When the douche is given by a single doucheur the preparation is the same as when there are two doucheurs. The patient is seated as before on a low chair. The doucheur, holding the tube which brings the water from the reservoir, pours it over the different limbs and the muscular parts of the body. The tube, which comes from a box containing the mixtures of water kept in the open air, is fixed to a particular instrument which ends with a watering apparatus, the water from which flows over the patient during the whole operation.

The local douche is given in a chamber or cabin divided into two compartments by a wooden partition, which has two holes bored through it so as to admit either the arm or the leg. The patient is seated on a low chair and passes his arm or his leg, as the case may be, through the orifice in the partition. The doucheur is in the other compartment, and he proceeds to massage the arm or the leg, or the affected part of the arm or leg, the thermal water flowing all the time, from a flexible tube connected with the reservoir, over the member under treatment.

In certain cases the chair used for the massage can be replaced by a particular stool; and under this stool is fixed a crooked tube which is joined to one of the reservoirs of the mineral waters and which ends in a watering spout. The jet of thermal water strikes the region of the perineum, and is of great use in the treatment of many cases of hæmorrhoids and of enlarged prostate. The name of *Corbin* has been given to this particular douche.

The *vapor* or *steam baths,* both local and general, are given in *bouillons,* or in the division of the *Berthollet apparatus.*

The name of *bouillons* has been given to cabinets which are connected with a certain number of douches. The mineral water rushes out bubbling from the apparatus which is used to produce this bubbling or boiling, and is converted into a pulverous, vaporous steam of 40 degrees to 43 degrees of heat. This vapor produces a rapid and abundant perspiration. The patient remains in this cabinet from three to ten minutes, the vapor bath being at once followed by a douche.

The apparatus called *Berthollet* constitutes another peculiarity of Aix. The daily use of this bath requires a consumption of 1,680,000 litres of water. A special arrangement of the Berthollet bath produces a mixture of air and vapor from the mineral water at a temperature of 44 degrees. This mixture is driven back into a receptacle of cast-iron, situated in the cabinet where the treatment is taken. The apparatus, which varies according to the part of the body to be treated, is fixed to the receptacle containing the vaporous steam, and is then directed to the affected parts, whether leg, arm, back, or shoulder. These different members of the body are first wrapped up in an impermeable cloth so that the currents of damp hot air fall directly on the parts to be treated. The treatment lasts about twenty minutes.

The Berthollet bath can also be taken in what is called a "box-bath." The vapor comes under the box, so that the whole body, except the head, is submitted to the action of the heat.

To the Berthollet is joined the *humage*. In the cabinet of this name are placed four *tambours* or *cuves*, low down, furnished with flexible tubes. The patient himself directs the current of air saturated with vapor to his throat, his nostrils, his ears, his face, or his hands.

The *baths* situated on the first floor are fed with cold and thermal water. Here the bathing is perfectly good and salutary. A certain number of the baths are connected with the douche cabinets, so that the bathing can be effected before or after the douche, without dressing.

The baths called "refrigerating" have eight cabinets in which baths may be taken in pure mineral water. The thermal water flows here as in all the other baths, but it is refrigerated gradually, and the cool temperature is due to a cool serpentine vessel through

which the water passes before reaching the bath. This mineralization is employed in certain *diathèsique manifestations* (morbid diathesis?) and in *dermotoses* (skin diseases?)

A small tube inserted in the tube of the thermal water serves to give the pressure of 45 degrees to the bath called "irrigation." It is a mediate douche resembling the *sous-marine* douche of Plombières. The patient, after he has gotten into the bath, directs the water-jet upon his body or on the affected part, either in full jet or in part; and, according to the layer of water between, the jet is more or less strong. The result of this simple process is wonderful. This last arrangement permits one to direct with great ease the mediate-jet on to the abdomen, and unites the effects of heat and gentle massage.

The *piscines* are fed by the same waters as the baths. The temperature is 35 degrees. In two piscines the temperature is lowered to 30 degrees Centigrade from ten to eleven o'clock in the morning. From a medical point of view, the piscine is very useful, as it permits the patients in certain cases (such as traumatism and arthritis), to move about in the water, movements which are otherwise difficult to make. The patient whose articulation has been immobilized is able to move about in the water, because the water bears him up. He is thus able to re-educate the movement of his limbs, and he learns again to walk.

The *physiological action* of the *thermal treatment* at Aix results from, or is caused by, many and divers agents. First, the physical and chemical factors are: the temperature of the water; its unctuousness; the abundance of the waters; a special electric action; its chemical composition, viz., its sulphurous contents. Secondly, factors resulting from the manner of applying the waters; massage; the mechanical action of the water; the action of the vapor or steam. Each of these various elements has its own peculiar action. The combinations of these various procedures produce different results.

The greater number of those who come to Aix have previously undergone various treatments, such as douches, baths, massage, and vapors. They come to ask and to look for a new treatment in the cure of Aix, which, happily, they find. This action is naturally complex.

The treatment of Aix-les-Bains, as has been proved by experiment, lowers the *arterial tension.*

The action of the treatment on the *nervous system* is stimulating as well as sedative. The effect produced depends on the manner of applying the treatment; such as temperature, duration, and frequency of the operations. We can obtain very different results, according to the method employed. The thermal fever which ancient authors were so pleased to describe, was only the consequence of the thermal operations too warmly and too frequently applied. That thermal fever was but a result of excessive zeal.

Nourishment is influenced by the treatment, as urological research has proved. The urine is rarified, and its toxic or poisonous power is increased. Phosphoric acid and the mineral elements generally are eliminated in smaller quantities; and at the same time we see the waste products of organic combustion, such as urea and uric acid, increase. In a word, the treatment has a mechanical action, both direct and indirect. The functions of the skin are stimulated by the flow to the periphery of blood, which is attracted by the warm water in contact with it; an abundant perspiration is produced, and an elimination is effected by this means. On the other hand, there is an acceleration of the lymphatic circulation, which favors the resolution and resorption of the exudates, effusions, and periarticular deposits. In a word, the diseased tissues tend to return to their normal condition.

The detailed manner in which we have described the treatment at Aix, and its effects, will allow us to make a statement, rather than a long description, of the *pathological conditions* which may suitably be treated by the waters of Aix-les-Bains.

The indication of the treatment of Aix must be considered every time we wish to accelerate nutrition. We must distinguish the principal indications from the secondary ones.

The principal indications are:

Chronic rheumatism in all its forms.

Rheumatism which deforms or disfigures, to which we may add the rheumatic nodes of Heberden and the rheumatoid arthritis of the English.

Muscular pains, lumbago, torticollis, and the various pains often called muscular rheumatism.

The sequelæ of articular rheumatism, both acute and subacute, in all its forms. The sequelæ of the various conditions called infective rheumatism.

Gonorrhœal rheumatism.

Asthenic gout.

Syphilis.

Every kind of neuralgia, and particularly sciatica.

Peripheral neuritis.

Chronic arthrites, joint stiffness, hydarthroses, post-traumatic, post-arthrites.

Muscular atrophy consecutive to an inflammatory condition, a nervous affection, or trauma.

The consequences of phlebitis.

In addition to these affections the treatment of Aix can ameliorate other states of illness (secondary indications) connected with nutrition, but the indication is then less absolute. Massage in conjunction with the douche suits certain cases of obesity, diabetic arthritis, and eczemas connected with the arthritic state. Hydrotherapy may be applied with benefit in most nervous affections. Many uterine maladies are relieved by copious irrigations, and the lavage is attended with great success in cases of mucomembranous enteritis.

No invalid ought to undergo the thermal treatment during or while just recovering from an acute attack of illness. This is one of the main *contraindications* for the use of the treatment. Tuberculosis and serious liver and kidney complaints are obstacles to the thermal treatment. Compensated valvular lesions of the heart (rheumatic endocarditis) can derive benefit from the treatment if well looked after. But all the other cardiac complaints must abstain completely. As to cases of arteriosclerosis, the cure of Aix is not denied them; only they must be well looked after by the doctors, who should exercise judgment and tact whilst having full control over the treatment.

The thermal treatment of Aix-les-Bains is perfectly suitable for the cure of *syphilis*. The sulphuration of the water in itself constitutes a specific medication. It is certain that the douche combined with massage is a valuable auxiliary to the iodide and mercurial medication. The thermal treatment acts, in the first place,

by its tonic effect; secondly, by aiding the absorptive and eliminating functions of the skin; thirdly, by stimulating metabolism. It also favors the passage of medicaments through the organism, by the successive formation of different albuminomercurials (by the absorption of the iodomercurial preparations and the elimination of the waste products). Thus the patient, a prey to an enfeebling and depressing malady, sees an amelioration of his general condition. He can also, without fear of accident, absorb a larger dose of the specific medicament, which, passing through the organism, acts potently.

At Aix-les-Bains, a specific cure for patients can be administered at different periods of the illness: old and new lesions, and most often tertiary lesions, are continually treated here. Inunction of large doses is often prescribed; we prefer, however, injections of soluble mercurial salts. For all these patients, the douche of Aix-les-Bains, with the *bouillon,* the vapor baths, and the profuse sweats which result therefrom, help the action of this specific medication, and allow of the continuous administration of a sufficient dose. This medication, more than any other, requires an attentive medical attendance.

The department of Mechanical Therapeutics is installed in a special pavilion, named the "Zander Institute." The Zander method consists in the use of the medical gymnastics devised by Ling, for which purpose mechanical apparatus is employed. Its principle is to graduate and localize the movements, to move each joint singly, to exercise each group of muscles one after the other, while measuring exactly the extent of each movement, and the energy of each and every effort.

In the Zander method, the resistance to be overcome by the patient's effort (which resistance is represented by the gymnast's hand) consists in a graduated lever upon which a weight can be moved. Compared with ordinary bodily exercise, such as sports or gymnastics, the mechano-therapeutic method offers many advantages, as it divides the work and diminishes the efforts. In the first place, during the movement the resistance accommodates itself to the natural variations of the result of muscular contraction. Secondly, the energy of the movement is measured exactly. Thirdly, the resistance to be overcome remains fixed. It is possible to

modify with exactness the energy of the movement, increasing or diminishing it, as the case may be. Different kinds of apparatus have been constructed, some for active movements, some for passive movements. Some of the appliances are made for mechanical procedures, such as vibrations, percussion, and kneading; others are orthopædic instruments. In the case of all these different appliances the doctor is sure to exercise the right muscles and not to touch the others; that is, he is sure not to touch those unaffected. He knows how to apply that amount of exercise which his experience has shown him to be the most useful; not to give more than is needed, and yet not to fall short of what is required.

The local results of mechanical therapeutics upon a single muscle, or on a group of muscles, and the mobilization of the corresponding articulation, may be immediate. There is an increase of the circulatory activity of the muscles, stimulation of the nerves, and increase of local heat and organic exchanges. Later on we can show that the repetition of the active movements brings about a favorable change in the muscular group—a sort of local dragging away. The muscular excitation acts mechanically upon the neighboring organs. There results from this excitation, at first, a transient over-activity of the system to which the muscles are joined; then a durable perfection of the functions.

Mechano-therapeutics have numerous indications. Only to suggest the most useful ones in the treatment of Aix, which give rapid results in all functional troubles, we mention but a few; namely, those which are the result of luxations, fractures, every sort of traumatism, and inflammatory or articular affections. Muscular atrophy and limited articular mobility may also be included.

The *water of Marlioz* is cold (14 degrees Centigrade), alkaline, and contains sulphur and iodine; it is much more sulphurous than the water of Aix (sulphur 0.263), and this allows of its use in cases which require a strong mineralization. The apparatus at Marlioz is perfectly installed. The treatment consists principally of cold inhalations; warm or cold pulverizations; local douches, such as those for the nose, mouth, throat, ear; and large sulphurous baths. The treatment of Marlioz is most valuable for bronchial catarrh; for chronic laryngitis and pharyngitis; for atrophic rhinitis; and in general for all nasal suppurations, as well

as for naso-pharyngeal complaints, and for external otitis. Besides these results, the baths of Marlioz waters, combined with their internal use, are most efficacious for children of the lymphatic and scrofulous type. In the radical cure of syphilis the water, taken as a beverage, is advantageously associated with the treatment of Aix-les-Bains.

It is a slightly mineralized water, feebly diuretic, analogous to the waters of Evian. Analysis shows that this water is perfectly suitable for internal treatment, as it aids the elimination of the substance formed in the tissues as the result of the diatheses.

The *treatment* of Aix is a powerful modifier of nutrition, and ought never to be undertaken without medical advice. Each and every case requires a special and different thermal operation, which must be chosen and applied accordingly. The mild and temperate climate of Aix allows one to commence the cure in early spring and to continue it till the late autumn. The months of June and September are generally the most agreeable, particularly for the cure. The duration of the treatment is from twenty to twenty-five days, allowing from sixteen to twenty douches. But this classic number in all thermal establishments is far from being absolute. Certain complaints require a more prolonged cure. It is sometimes advantageous to make two cures, separated by an interval of rest more or less prolonged. As Despine remarked, "The waters of Aix, like so many other medical means of curing, cannot be administered to all in like manner, nor in the same proportions."

The patient must avoid all excess, no matter what it may be. Late hours, too abundant food, the use of alcoholic beverages, even those cordials under the name of "tonics." Exercise in the open air, a walk or a drive, is always good, provided it is not carried to fatigue. One must guard against catching cold, remembering that the treatment exposes one more easily to the liability of taking cold, as one is more sensitive to the action of air or draughts. Nothing is so good in this respect as the wearing of woolen garments next to the skin. Even when the cure is terminated, the patient, though he may not feel tired or fatigued, is in need of rest—hygienic rest; and to this end a sojourn up the mountains, or simply in the country, is useful if not necessary.

MANAGEMENT OF EXHAUSTION STATES IN MEN

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The problem of the overwrought man of affairs is by no means a simple one. It is constantly recurring, demanding of the medical adviser wisdom, tact and a practical familiarity with all the measures recommended, the regions to be visited and the forms of life prescribed. Many of my earlier years, spent as the assistant of a great neurologist, taught me the paramount importance of this personal knowledge; giving me also a wealth of experience obtainable in almost no other way. Much of this was gained by traveling extensively as the physician-in-charge, thus supplying a working knowledge of practical details.

Given, then, an overtaxed, busy professional man, a captain of industry, a statesman or scholar, exhibiting the well-known phenomena of physiologic irritability with fatigue, how shall we proceed to achieve the quickest, the most permanent repair?

Our resources group themselves under five heads, subject to many variations, combinations and adjustments:

(1) We may permit a continuance of occupations, but must modify them. We may employ partial rest, enforce relief from strains, begin the education of the spirit, regulate life, diet, sleep and activities; use medication, sedatives, tonics, digestants, eliminants, rational auxiliary measures, hydrotherapy, open-air life and the like.

(2) We may employ occupation and educational therapeutic measures. These are exceedingly valuable, but far too little used; because, to employ them, the adviser must himself supply large resources and possess wisdom. Much time and companionship are demanded of the physician. An extraordinary nurse or attendant, preferably a young medical man, will be needed as a supplement.

(3) We may need to use absolute rest for varying periods. This should be not less than a month or six weeks, preferably eight, followed by change of scene, prolonged absence from responsibili-

ties and relief from demands on attention. This may include spa treatment, residence in special climates and maintenance of conditions antithetic to the customary.

(4) Sanitorium treatment may be useful as a makeshift, an inexpensive, wholesome form of routine which suffices for plastic, bidable men.

(5) Exercise and discipline cures: These accomplish extraordinary results in some cases, and are, when permissible, most economic of time.

Take a familiar instance illustrative of a peril from which many do not, or will not, escape. It may prove suggestive to review our findings and the patient's reasonings, to point out his objections and outline our recommendations.

A successful men of affairs, near middle life, states that he recognizes himself to be miserably tired, painfully wearied. He can no longer fix his attention on even simple problems, or maintain interest in those matters which constitute his life and are ordinarily pleasurable. He becomes readily confused, peevish, loses his temper, explodes absurdly, is painfully aware of, yet laments, his ill-judged actions. Perhaps he sleeps badly, waking in the small hours, vastly worried, or in the early morning, conceiving himself to be beset with calamitous menacings. His nutritive functions may be well maintained, though they are more often sadly out of gear. He admits a pain or heavy feeling at the nape of the neck, enjoys resting the back of his head frequently on some convenient surface, a characteristic feature of brain fag. He pleads guilty to inadequate or no vacations—he "has n't needed them;" or what time he has spent away from business has been in an atmosphere of jangle and glitter selected by his wife or daughter. This last form of complaisance has destroyed many a useful man, his financial, his moral or his physical integrity. Or worse yet, he is a "man who knows his own mind." When he leaves his business he "is not going to immure himself in some pokey hole; he needs amusement—life." He has had over much life and now retains only a feeble tenure on what remains of it. Dr. Osler in a consultation concerning a man who described himself as a "plunger," remarked in his droll way, "quite so, one plunge more and it will be into the pit."

The whole condition may be due to the effects of unending routine, minor or major anxieties, meeting continuously a succession of fixed engagements, giving undivided personal attention to details of business, family or charities. Fixed attention, when it is long maintained, induces exceptionally complete nervous exhaustion.

It remains for the adviser to decide if it be permissible to accede to the often dominant request for " a tonic to put him on his feet "—there are " a number of pressing matters which demand attention." There always are. If he dies, however, another will take them on, well or ill. Is his life worth anything to him? Yes. He can be patched up, of course. The penalty of half measures is a greatly diminished efficiency, and for all time. By the exercise of great wisdom it is permissible to compromise, but always with vast risk,—to temporize, curtail responsibilities, lessen the routine demands, use partial rest, regulate the life judiciously and yet permit a continuance in the traces. Too often the physician is deceived as to the gravity of the situation. A Circean webb of difficulties is all the time weaving about the wavering judgment of the sufferer. The old-time clarity of decision shrinks. Mistakes are made, false steps are taken to repair errors, chagrin follows; secretiveness is thereby developed, and, sooner or later, the physical and psychical conditions are found to be far worse than before. Business reputation, even character, has already suffered. The problem presented is thereupon a hundred fold more complex. Few physicians, perhaps none, can unravel it. There is one course left; even that is far less feasible than before, but it must be unfalteringly taken. Radical measures are demanded.

The man is exhausted now; hitherto he was only tired out but still capable of full repair. Had he been earlier divorced completely from his surroundings, his sources of exhaustion removed, had he taken full opportunity to regain tone, by the end of three or six months he would have been found as good as ever, indeed, perhaps better than before. For, with renewed energies, the product of rest, change of scene, the broadening influence of travel, comparisons of view with new acquaintances, contact with strange people (so like ourselves yet with differences highly instructive), he would be enlightened and refreshed. Thus renewed, at the

zenith of his powers, a man returns from roaming an incalculably better citizen, a wiser father, a shrewder man of affairs. A six months' trip to such a man is the full equivalent of a handsome life insurance. It is a practical annuity for his family.

However, if the disastrous stage above described is reached by the combined blundering of himself and his physician, the consequences must be faced and suitable remedies applied. A complete breakdown demands absolute rest, full isolation, protracted nutritive repair, not less than two months in bed, followed by two years of careful living (see articles by author, "Repair of Will Loss," International Clinics, vol. ii, seventh series; also "The Conduct of Rest Treatment," vol. ii, thirteenth series. Mind and body are now seriously impaired. They may be rejuvenated, but never fully restored; 60 to 75 per cent. restoration is all that can be then expected.

Where the fundamental fault is wholly or mainly due to deviations in original functional competence, the problem is to measure possible progress by the standard of the weakest organ; to conserve the action of this one and supplement it by bringing the others in line, and maintaining an even plane of advance toward the normal for that individual. When the basis of disability is exhaustion in the central nervous system, and where this has progressed to a point demanding rest, then this rest must be absolute of mind and body for a time, otherwise the nutritive forces cannot be rehabilitated. The most significant data in the differentiation are psychical. Experience, a wide familiarity with analogous states, can alone fit one to estimate correctly and control the derangements.

A few points from a full experience may help here. On being put to bed, isolated from family, mail, newspapers, business, etc., there is usually some revolt: "his temperament is peculiar, this sort of thing will do him harm rather than good." "It may do for some but not for him; he will be made worse, not better. He can't be well without exercise, can't eat, can't sleep, can't have his natural movements, etc., etc." As a matter of fact, after a week of bed a sense of "heavenly peace" replaces the peevish, flickering energies. After two or three weeks in bed, few exhausted men can be induced to budge. After six or eight weeks, if so much absolute

rest is necessary, something of a struggle is involved to get him up and about. Yet "he feels perfectly strong, could run around the block; could resume his work as well as ever, etc." The first sitting up, which should be for only ten or fifteen minutes, is found to be quite enough. An amazing slackness of the joints is admitted; the "head spins." Often a sickening sense of weariness is experienced, especially if the erect position be persisted in long. The upsittings may be increased from once to twice daily and by five minutes only, longer each time till an hour, forenoon and afternoon, is thus spent. It requires two or three weeks gradually to resume activities—perhaps double that time. A most vital point, one that tests the skill of the physician, is to regulate the resumption of motor activities (see paper by author, "Motor Education" in "Convalescence in Invalid States," *Medicine,* September, 1905).

The surest, most economic, and permanent repair of the majority of the conditions mentioned is gained by change of scene, especially a return to primitive occupations and forms of life. This is often necessary, also, as a supplement to other measures employed. The first requisite is to spend enough time in moving about; it should rarely be less than three months, better six. The places that should be visited will depend on the peculiarities of the individual, his personal habits, tastes, previous opportunities. When a man has heretofore taken fair vacations, has travelled much, seen the wonders of the world, his consciousness being jaded by much variety (however injudiciously employed) by far the best course is then to "hit the long trail." This may be to plunge into the "north woods," or travelling by canoe, paddling and poling, "making portage," carrying packs, aiding honestly in the day's duties, "rustling for grub," making fires, cooking, "cutting boughs," always working or loafing from sun to sun. Thus is gained that power, which Anteus showed to Hercules by repeated embraces of Mother Earth. Or let him take the pony trail on the plains or Rocky Mountain foot hills; explore Zuni remains, Navajo villages, "box canyons," cross what remains of the American Desert, or traverse the "bad lands"—"Mauvaises terres pour traverser"—not so bad as the name would imply. These open spaces are full of delight , canyons, buttes, "mesas" and wide grassy areas where once ranged much big game. The author has been blessed by such opportunities, enjoyed all

these delicious rejuvenating influences, which afforded him as much, perhaps more, benefit than the patients with whom he journeyed.

To regain youth, pristine capacities to eat mere "food" with joy and thankfulness, to sleep as the just sleep, to wake with the light, singing "Te Deum," to swim in waters far away, to commune with the "little people of the wild," to acquire muscles of steel, nerves of compelling power, endurance of the thoroughbred horse —all this is achieved "on the long trail." A week or two is good— a mere foretaste, a preparation. It may do for a man who is merely tired. To repair exhausted energies, especially those of middle life, due to long strains, nerves jaded by years in harness, demands many weeks, really, three to six months. To one who never experienced the woods or the "great open" the first week may disgust. Compared with a house a tent, a shack, especially if it rains or snows, creates a great longing for the comforts of home. It is needful for the "bear leader" to keep a tight hold on the consciousness of the soft weakling; much cajolery, encouragement, flattery or frank argument will be called into play. The toughest proposition in my experience was a melancholic lawyer of endless pertinacity and sophistry who frankly "detested all camping." Him I "held with my glittering eye" till we were too far away for him to return alone, as he bravely and repeatedly tried to do. After a month of hardening he forgot home, office, investments, and became so strong, to my intense surprise, that his feats of walking, his canoe paddling, his voracity for "hog and hominy" surpassed belief.

Years afterward, myself in the woods with a new guide, talk arose about tenderfoot wonders and tales were related about a certain "lawyer who almost wore him out." Suddenly producing a note from this man it proved to be my whilom patient, who had acquired a taste for the trail and had achieved traditional valor all his own with my woods runner. The "coureur des bois" is still what he was in the pioneer days. Emulate him and it does not take long to secure his attributes; endless endurance, a wide confidence in oneself, a serenity of mind useful in any sphere of life, above all, cheerfulness and self-control through good and evil report.

Much wholesome philosophy can be thus acquired, a clarity of

vision, a perspective on one's peculiarities and characteristics, a capacity to think steadily on one line, to be happy with the least of human necessities, which qualifies one to call oneself a man, and, what is more, to do a man's work. It is doubtful if these attributes arrive at perfection by any other course. In every tired man the good is bound up with inevitable evil. A jaded spirit is no longer sane, no longer clear sighted. Morality shrinks from overmastering temptations. The wood's life can cure all this. Indeed, it is difficult to see how a man can attain genuine good health unless at times he goes afar, communes with nature for a time, and reacquires some part of his lost animal inheritance. In these long vistas one can best get that perspective on oneself which brings about equanimity.

Change of air, especially of companionship, is needed by everyone, well or ill, not because the air one ordinarily breathes is inferior to that of some other place. Monotonous labor, wearisome routine, continued fixed attention on affairs, themselves trifling, exhausts the fountains of force as surely as the sparks fly upward. The matter of climate is not so significant as is popularly believed. Few climates are so bad at any time as to require escaping. The dulling effect of humdrum is, however, most disastrous, especially upon certain temperaments. Not only does it limit zest, buoyancy of spirit, mental clarity; it impairs the moral sense, wears out the niceties of differentiation; but be it noted, does not permit growth in potencies, in judgment, in breadth of grasp. A man should aim to arrive at relative perfection, consonant with his inherent powers, toward late middle life. Monotony in labor fosters none of these perfectabilities; it distinctly inhibits them. Normal development, reasonable achievement of expectations, rarely come to the patient drudge. Even maintenance of mental integrity is thereby severely warped, impeded or damaged. While much may be said in favor of the effects of a life of peace and material prosperity, yet the growth of communities and nations is always enhanced by the stimulation of opposition, of difficulties met and overcome. Resourcefulness is only developed in the face of perils, of difficulties and opposing forces to be met and vanquished. The pioneer is always a model. To live his life, if only temporarily, makes for the best manhood, the highest character and capacities.

SANITORIA; SPAS; REST CURES

Sanitorium treatment, spa methods, conventionalized systems so largely developed in Europe and growing in favor here, are all to be commended on one ground or another. They all supply one important need, viz., regularity, simplicity, control of diet, of activities, along with less or more of various useful measures, the specialty of the place or the physicians in control. They all make for the essential prerequisite, obedience to routine, self restraint. Socrates enunciated this last as the one chief desideratum, "self restraint."

All these organized expedients are valuable for certain conditions, temperaments, stages of progress. They exert a restful effect, tend to readjust the overtense mental attitudes, furnish novel, unaccustomed outlets for primal energies. Unfortunately in sanitoria there is too much of association with sufferers of like kind, too much of mutual experiences to be interchanged, talk, fighting curative battles over again, self pity, criticism of methods, etc. (See "Repair of Will Loss," by author, INTERNATIONAL CLINICS, vol. ii, seventh series.)

The chief objection to these routine "cures" is that sometimes a patient acquires the "sanitorium habit," a mental flabbiness, overmuch soltitude for minor ailments, a conviction that "the constitution is peculiar;" one rift in the lute suggests another, the patching up process becomes a career. Among these, I have had some success in turning the pupil into a teacher equipped with the first principles of "new thought" or "mental culture" and send them forth to grow and to proselyte.

EDUCATIONAL AND CORRECTIVE EXERCISES

As a part of all measures designed to repair exhaustion states educational and corrective exercises seem to me essential in achieving the best results. At the outset it will be evident that exhaustion states invariably arise in, they may be said to be caused by, erroneous, heterogenous expenditures of force. It is seldom, perhaps never, that the prodigalities of energizing are along normal lines, conserved by accuracy and judgment.

My experience is large enough to serve as a basis of positive

opinions. Men like Weir Mitchell, Horatio C. Wood, with whom I have long co-operated, have repeatedly asserted in my hearing what has become accepted as an aphorism, viz., "It is not brain work but worry which exhausts." In the field of athletics also, where much of my experience was gained, I long ago recorded the aphorism that "no entirely sound man is damaged in athletic competitions by however so earnest strivings while competing in an event for which he is adequately trained." To be sure the element of morbid excitement, loss of psychic balance, disorder of emotion, whatever disenthrones judgment, may contribute to bring about physical catastrophe. The exhaustion states most serious in men are chiefly of the fountain of force, not of the motor machinery, though they are closely interdependent. I have repeatedly examined athletes, as, for example, at the end of a "six days' go-as-you-please" race and those who maintained "equanimitas" and poise were not damaged.

An individual is capable of generating and appropriating so much of nerve force, utilizable energy, and no more. When the centres are called upon to respond beyond their ability, are kept at too high a tension, the result is disturbance somewhere. For example, the function of vision, even when performed under the easiest circumstances, through correct glasses, but in excess, unless the eye and its appendages be absolutely normal, must become exhausted, and thereby the whole organism suffers. When irritability is evidenced, conservation is demanded or the integrity of the organism may be shattered. From this starting point not only may serious happenings directly arise but weakened organs collapse, *i.e.*, lungs, heart, kidneys.

Among the most efficacious of measures here is long range vision for hours, freedom from all strain, even that of talking to companions. This is practically educative exercise of the eye.

Exercise is use. Where misuse has perverted function nonuse alone is never relief enough, except temporarily. The cure is accurate use. Every one beyond early adult life is the better citizen, the healthier, happier member of family or community, who, by accident or choice, is induced to make right, regular use of all functions.

Disuse is as hurtful as misuse. The most potent cure of

ailments is correct consistent use, exercise. From this truth there is no escape. Sophistries are here perilous. It is true we occasionally see instances of apparent controversion in this sphere.

Some people appear to live healthily and contentedly without making right use of their minds or bodies. Upon examination, their lives will be revealed as mere existences. They do not live, they cumber the earth. They accomplish nothing.

The exercise of the mind, the spirit, thinking good thoughts, doing things naturally, wholesomely, kindly, helpfully, may suffice to maintain fairly uniform health. Conversely, overdoing unwisely, as, for example, busy persons driving themselves through misplaced zeal to take violent exercise when fatigued, jaded, irritable, from faulty methods of work, may and often do fall into evil plights. Always it is desirable to take counsel from some one who is equipped by knowledge and judgment to guide wisely. Nowhere, in the field of human economics, especially in the realm of physical repair, is there greater need of judicious expert advice than in the matter of motor education. (See article by the author, "Motor Education in Convalescence and Exhaustion States," *Medicine,* September, 1905.)

This subject is too large for more than allusions here. Suffice it is to say that that physician is most capable of securing fullest repair, rejuvenation of completest efficiency, as well as developing latent perfectabilities, who guides his patient discreetly in the direction of his gross motor energies. Inhibition is as important as stimulation. Permit me a brief summary of the points to be considered. Latent weaknesses exist in every one, resultants of defective development. These may, or may not, have been revealed by the phenomena of the lowered general condition. They must be searched out, estimated, corrected, trained. This can be done by regulating the organic activities, inducing uniformity in vegetative functions detecting and correcting eliminative faults. Among the most forceful is manual treatment of the viscero-motor centres, establishment of the vasotonus through regulation of the nutrition in the governing centres in the cord. Next in order is the correction of prolapses, ptoses, displacements of the viscera. (See article by the author, "Rational Treatment of the Visceral Ptoses," *New York and Philadelphia Med. Jour.,* Aug. 4, 1906.) Third

is the searching out of contractures in the supporting structures, the skeletal muscles, the ligaments, etc. The thorax especially demands attention, its mobility, cubic capacity, so essential to the activity of the contained organs, the heart, lungs, etc. The tissues of the neck, their degrees of elasticity, exert vast influence for good upon the organs of special sense, the circulation of the brain. Unless the return currents of blood and lymph are unimpeded, their precious tissues suffer. All physicians know how influential the integrity of these is upon the status quo. Contractures in the ligaments of the vertebral column are deeply significant. Perfect flexibility in the intervertebral tissues makes for uniform circulation in the segments of the cord and this for vaso-motor, hence vegetative competence. (See article by the author, "Conservation of Energy in Ageing Tissues," *Pop. Sci. Monthly,* March, April and May, 1904.)

Finally we come to a consideration of the great engines of oxidation, the big muscles. This subject, muscular training, needs the space of a separate paper. (See article by the author, "Physical Economics," *Med. News,* 1904.)

It will be plain to any one who can appreciate the potentialities in the outline submitted, that attention to these factors must influence profoundly the vegetative processes.

EXERCISE AND DISCIPLINE CURES

Experience is often a better teacher than the best of inductive science. Many forms of "cure," successful in diverse maladies, would seem, at first sight, too onerous or perilous for a specific case. However, bold, confident empirics often arise, showing us of the "learned profession," that our cautions lead sometimes to error, that timidity may prevent good results. An extreme type of an excellent procedure, accomplishing vastly more than the medical mind would ordinarily pronounce possible, is the method for years employed by Mr. William Muldoon (White Plains, N. Y.) of "putting men into condition." This is especially interesting and suggestive to me, confirming many beliefs long held. His methods are capable of modification and improvement for application to delicate folk by the physician. The course of treatment consists of alternations of free exercise and rest, under his direct supervision; regulation

of diet, bathing, and above all, enforcement of absolute, implicit obedience, a rigid routine, almost a military system. All this makes for moral and physical regeneration. The time occupied is seldom more than three to six weeks. Those who undergo this course are, as a rule, men originally fairly robust, yet out of repair, due to a wide variety of derangements, the common basis being neglect of the fundamental principles of personal hygiene. These men are mainly exhausted by mental work, devotion to business, professional work, and especially by over-indulgences in what are catalogued as "the pleasures of life."

Mr. Muldoon is to-day, at sixty-three years of age, the epitome and prototype of the vigorous man, capable of, and exhibiting daily, the most strenuous action; sustaining large responsibilities, maintaining absolute control of himself and other men, themselves among the most successful and dominant in all departments. He began life as a professional wrestler, achieving the highest position, an unbeaten champion, and holding this far beyond the usual age. My recollections of him are vivid and inspiring. He was, and is, the perfection of conformation and action, splendid, impressive, dignified, of limitless courage, resource, judgment, swiftness of decision, motor accuracy and self control.

Happily for many of mankind, he turned his energies in early middle life to teaching others, training champion athletes and, later, curing of their ailments those in physical or mental decrepitude by means of unique measures, and with amazing success.

He has equipped a country place, well and wholesomely situated, with a large gymnasium or exercise hall, shower baths, etc., but no mechanical apparatus. Here he takes his pupils and puts them through judiciously adapted activities. The day begins before eating, with the play of "medicine ball," a large sphere the size of a watermelon, weighing from three pounds upward. This is tossed from one to another, caught and thrown back, upon a system modified for different capacities and gradations of vigor.

This is followed by a cool shower bath, 70 to 80 degrees F., regulated as to time, distribution of impact, method of soaping and subsequent hand rubbing. Later in the day, all ride horses for an hour or more, usually at a walk or gentle trot, increased to a thorough jolting, alternated by a walk on foot. The water which

is drunk after all hard work is hot, never cold. The day begins early, activities are followed by rest in the open air, carefully selected meals, followed by rest; a minimum of tobacco, no alcohol; billiards are encouraged; no naps, but early to bed.

It might appear that this course would overtax many who are unused even to the most moderate exercise; who are so entirely "out of condition" as to exhibit feeble heart muscles, organic fatty infiltrations, general flaccidities. Dangers there are from concealed arteriosclerosis, kidney defects, visceral ptoses, relaxed omenta, impaired vaso tonus, etc. Nevertheless, while these may seem imminent, yet I have never heard of any positive damage resulting. All those whom I have questioned who have taken this course, and they are many, are enthusiastic as to the beneficent results. Some were often greatly vexed at the severity of the treatment, physical and psychical, the stern absolutism, the bullying, etc., nevertheless, no one could fail to admire the consistency, the reasonableness, the wholesomeness of such a period of return to a fundamental plane of intelligent animalism. Intelligence needs curbing and guiding when it gets off the track; it becomes too transcendental; equally does animalism need to be clubbed into shape if the man is dominated by his lower centres. The methods useful for each have many points of contact.

The chief purpose of recommending exercise for exhausted men is not in accord with popular notions. Much of the satirical objections urged against "active exercises," "physical culture," "monkey stunts," is perfectly sound, although too often overstated. The exceeding great value of judiciously regulated exercises is beyond question; it is applicable to all and will be carefully formulated later. Many assert that if exercise is needed at all it can be taken profitably in the course of one's daily duties. It is claimed by many women that they could get all they need by sharing in household labors; that men could secure enough while walking or in the pursuit of business and the like. This is true, but only in part. Motion is always of value, aiding expression, translating thought into manifestations of external force; also in maintaining habits of symmetrical action and reaction. If this were done adequately, accurately, consistently, all might be well. But how much of this is consistently done by most of us? For instance,

a man assures us that he wearies himself overmuch in the day by standing long, moving about in his business; or a woman derides the idea of more exercise when she exhausts herself continuously in domestic duties. It is true they are better for this movement than if they remained entirely sedentary, but the usual claim is that all this merely induces weariness; they would welcome emancipations rather than undertake more. Certain facts will become apparent on scrutiny. Exercise thus obtained is really a labor, a monotony. Lax motor habits are thus formed, born of the necessity to put forth the least effort to accomplish compulsory results. Accuracy is only achieved by economizing force. The motions involved in manual labor are restricted, overtiring, failing to bring into play accessory parts, such as the supporting structures, which are held overlong in tension, involving undue pressure on feet or back, often followed by minor painfulness.

The real object of exercise is to achieve uniform, accurate adjustments, capacity of full stretchings, elasticizing of parts ordinarily not employed; always striving to secure sympathetic responses of the centres of intelligence, especially motor centers. When this facility is lost, or out of repair, a large array of minor disorders inevitably arise which tend to grow worse. In consequence of this a most serious fault results, loss of uniform interreaction between governing centres and the outlying parts, inducing fatigue rather than repair. Fatigue is a pathological condition, which, if continued, tends to impair vitality, causing, also, local damage.

THE CRUX OF THE WHOLE MATTER

Finally, let me preach a small sermon. I shall borrow thoughts,—we all do. But now take an excellent text, the very words of which are filled with significance for the world-weary man: "They that wait upon the Lord shall renew their strength" (Isaiah). You observe, strength is promised those who "wait." Milton, quoting unconsciously half remembered thoughts credited by scholars to old philosophies dimly sketched far up the aisles of time, said: "They also serve who only stand and wait." Tennyson sang: "Time, the great healer, unto thee I lift my hands, my heart, my head." All the practical recommendations suggested in the foregoing pages are merely devices to seize and make one's

own the real curative agency, time, rest, cellular readjustment. The serene point of view is obtainable only by holding the spirit in equipoise; by letting slip the shackles of hurry, by anchoring fast to the one greatest thing, "peace." One may diligently pursue each and every measure, rational, suitable, eminently indicated though they be, to recover the dissociation created in him, but, unless through and by it all he achieves a central spirit vision, equanimitas, they will leave but a fleeting impress on the soul. Each man, I take it, will admit that his sincere desire is to acquire full control over his powers, so that they may be economically expended for pleasure or profit. Forces work efficiently only when unhurried. Power and dominion can only be put forth to accomplishment from the habitual standpoint of deliberation. Accuracy is conditional upon following the rule of motor efficiency laid down by me so often in teaching muscular co-ordination, viz., begin each act from the standpoint of complete relaxation, carry it onward by steady increments of force to the point of fullest tension. It is a parabolic curve. Thus, and thus only, does the fencer strike precisely where and when he decides to hit. By the various means outlined one may secure the habit of accurate deliberation. Unless this attitude of mind is gained, or regained, all effort is a failure. The blessed woods, the glories of the open, the breadth—the immensity of the sea, all should teach the lesson of suppressed, concentrated power. Speech is desirable, perhaps necessary, but communion with our higher selves is better.

Talk, conversation, invites diffusion, leads to uncertainties. Action is always marred by overmuch refinement in choice. Growth of intellect, of character, of worldly accomplishment comes not from crying aloud, "how shall I do this or that?" Thought, leading to decisions, proceeds best from the closed chambers of the mind. Time is an essential factor,—be it much or little, it must be enough.

Strength comes from soltitude, a waiting, a communion with the best in us, which is at one with the divine spark. With strength comes judgment, and above all, clarity. The body, when exhausted, must be provided with opportunities for renewals, but the lesson of life is best learned, the power is only achieved, by a knowledge of our own potentialities and limitations.

Medicine

A PLEA FOR LAPAROTOMY RATHER THAN PARACENTESIS IN ASCITES

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Notwithstanding great advances in the knowledge of abdominal diseases and their treatment, we see frequent evidence of treatment in actual patients without regard to the nature of the underlying disease. The term ascites is a convenient one, and, if used symptomatically, with the purpose of finding its cause, and carrying out proper treatment for that, no harm can follow. But we frequently see cases in which the diagnosis stops with the demonstration of free fluid in the abdomen. This is certainly a difficult task in many cases, but never the only one.

In other cases, following the detection of ascites, the fluid is taken away in order to make more certain the palpation of the abdomen. If the existence of a local disease is then made out, and proper treatment begun, there can be no reasonable objection to the plan. But if the examination reveals nothing more than was known before, and only symptomatic treatment is carried out, a grave error may be committed.

Sometimes the fluid is subjected to subtle chemical or cytological or bacteriological investigations. From any or all of them many interesting things may be learned, but I question if they are worth the time devoted to them, unless speedily positive, and I suspect that more bad than good diagnoses are made through such examinations. At the best, they never reveal enough. Cytological examinations may demonstrate tuberculosis or carcinoma; or inoculations may disclose tuberculosis of the peritoneum, but the main question for the thera-

peutist is not whether there is cancer or tuberculosis, but where, and how much, cancer or tuberculosis exists, in order to determine the treatment.

One should make the most exact possible diagnosis, using paracentesis and refined technical methods as far as convenient, but unless these give absolutely final results—which they rarely do—it would be better to open the abdomen, let out any fluid present and make a complete exploration with the express purpose of finishing at once any operation indicated and feasible. The situation, of course, should have been fully explained to the patient. I am well aware of the difficulties, dangers and fallacies of this method, but I maintain it is better than the less radical one. The examination must be thorough, and should be based on what has been learned by other methods of exploration. In chronic cases the fallacies and difficulties are not so great as in acute ones, or as in chronic cases with acute symptoms, such as obstruction or peritonitis.

As an example of this I mention a case that I was fortunate enough to see in the clinic of a surgeon whose name, if I mentioned it, would be recognized as that of one of the greatest abdominal surgeons Europe ever produced. A man was brought to his clinic with acute intestinal obstruction. At the laparotomy nothing could be found to account for it, unless possibly a long saccule of the ascending colon, which it was thought might have caused volvulus or kinking. The saccule was accordingly excised. The patient died in a few hours and was then found to have a circular carcinoma of the descending colon below the spleen, the size of a large orange, and no doubt easy to feel before the acute symptoms appeared. Such an experience not only shows the value of careful examinations in all cases without operation, but the necessity of a full examination at an operation, unless a satisfactory cause is found or the condition of the patient forbids it.

Every now and then my attention is called to this subject by meeting patients with ascites who have been treated symptomatically, and my efforts at more radical treatment have often failed because of a widespread belief in the propriety or efficacy of paracetesis or some internal medication in such diseases. I have learned that it is one of the easiest things in the world to obtain permission to tap an abdomen, easier than some seem to find to use a stomach

tube, and I have also learned how detrimental the early use of the trocar may be to radical cure.

It is known even to the laity that many diseases of the pelvic organs in women are surgical and may generally be healed by operative treatment. But if the disease is masked by ascites, unless the patient goes to an operator for the specific purpose of an operation, such a suggestion is likely to be declined, and tapping or drugs are used, until it is too late for anything better.

How much a patient can stand in the way of tapping is shown alike by the annals of surgery and by graveyard epitaphs, and I shall mention a fairly good case myself. As an example of persistence in drug treatment I might mention one of my patients who used Hay's method with strong doses of Epsom salts for ascites, during eight months, every other day.

Out of a larger number of cases I select the following, as illustrations of what is sometimes done and what may be done in some diseases associated with ascites.

Case I.—Malignant teratoma with ascites as the earliest symptom, two years before the diagnosis was made.

L. G., æt. sixteen. (January 25, 1895.) Enlargement of the abdomen became noticeable soon after the patient spent a night dancing, four months before admission. At first the swelling diminished under medical treatment, but for three months the enlargement has increased.

Symptoms and physical examination reveal no disease except that in the abdomen. Examination of abdomen shows moderate general enlargement, with bulging in the sides. There is movable dulness and distinct fluctuation. As the patient would not enter the hospital and had to return home by a long sleigh ride, I refused to aspirate in order to palpate the abdomen more thoroughly, and did not make a pelvic examination, anæsthesia not being praeticable, for obvious reasons, but made a tentative diagnosis of either tuberculous peritonitis or disease of some pelvic organ, and recommended laparotomy for diagnosis and treatment.

October 29, 1895, the patient returned, having been treated with drugs in the meantime. This time a mass could be felt in the left side, about the size of a cocoanut, hard, not elastic. Smaller masses extended to the pubes. The general condition, including blood,

negative. The diagnosis of tumor of ovarian origin was made, with ascites secondary; tuberculosis excluded for reasons not necessary to repeat. The long course without positive signs of malignancy was considered promising. The patient was advised to undergo an operation and warned against tapping.

Some time after that an operation was performed in another hospital. It was reported that a dermoid cyst of the ovary was found and removed.

The further history shows the importance of careful examination of the patient and expert examination of the specimen removed. Some months after this operation, another mass appeared, involving the adjacent tissues widely. Examination of a bit excised per vaginam, sent to the Pathological Laboratory of the University for diagnosis, November 5, 1896, revealed a malignant teratoma. It can hardly be doubted that an operation at an early period would have been much more likely to prevent recurrence; but in this case, as in so many others, an impression had been made by the seeming success of the early treatment by medicines. Active catharsis or diuresis often make such patients think they can avoid operation if only they find a doctor who can prescribe a remedy as efficient as they had before.

A still more instructive case is that of Mrs. H. S., æt. sixty, admitted to the medical clinic on account of ascites of four years standing March 5, 1902. "The dropsy began with soreness across the abdomen, ascribed to a fall some months before. Nine months after the soreness an enlargement began; she was tapped and 15 quarts of clear yellow fluid removed. Since then she has been tapped 64 times, with an average removal of 12 quarts of fluid each time. It is now necessary to tap every two weeks." Pain in the back and a chill follow each tapping, but no effort seems to have been made to perform a more radical operation. Much "strong medicine" was used, leading finally to complete loss of appetite, vomiting, severe hiccoughs lasting ten days, and extreme emaciation.

There was a history of biliary colic, with occasional jaundice and slight intermittent fever. This was ascribed to typhoid fever which the patient had in 1866, followed by jaundice. A year after the abdomen began to swell œdema of the legs appeared and has continued. Sometimes there was temporary œdema of the face.

There have been severe attacks of pain in the heart region, almost anginose in character, with dyspnœa. An abstract of the notes says:

"On admission, the abdomen is enlarged, about four inches above the level of the ribs; bulging at the sides. There is dulness moving with change of position and reaching three fingerbreadths above the navel on sitting up. Below the navel an uneven mass can be felt, extending down and to the left to Poupart's ligament. The patient says she has known of this for one year, but for a longer period has been aware of 'bunches' in the abdomen.

"All the thoracic organs are pushed up. In the sitting position the apex beat is in the fifth interspace in the nipple line. The cardiac dulness from the fourth rib to the nipple line and the left edge of the sternum. There is a soft short systolic murmur, loudest at the second right costal cartilage, transmitted up in the neck. No peripheral pulsations. Radial pulse of fair size, soft, not quick.

"Blood: Red corpuscles, 3,222,000; leucocytes, 7,385; hæmoglobin, 60 per cent.

"Urine: Negative."

The ascites had been thought by many physicians to be cardiac, but from the course of the symptoms I thought it more likely the cause was to be found in the pelvic tumor.

A diagnosis of fibroid tumor of the ovary was made and the patient referred to the gynæcological clinic, where Dr. Reuben Peterson operated March 14, 1902. The large irregular mass of fibroid tumor was removed and the patient made a good recovery, complicated by the passage of gall-stones.

She then remained well for nearly four years and died suddenly, apparently from the old heart lesion.

In the case just cited the relief from tapping was very brief and the operation had to be repeated often, but still that method, with drugs, was followed for a long time. In the next case the relief given by a single diagnostic tapping kept the patient from further treatment for many months.

Ascites for more than three years. No cause discovered until the end of that time, when a large ovarian fibroid was discovered. Operation. Recovery.

Mrs. W., widow, æt. forty-seven, admitted September 13, 1905. Family history negative. Patient was married eleven years.

Never pregnant. Menopause at thirty-nine. Has passed a good deal of mucus in strings and cylinders with the stools.

Four years ago began to have pain in stomach region. Three years ago the abdomen began to swell and soon reached its present size. Two years ago the legs swelled.

Examination on admission showed general emaciation, but no notable changes except those in the abdomen, as follows: "The abdomen is large, measuring 47½ inches; bulging in the sides. Veins on the hips and sides of abdomen distended; not those in the middle part. Movable dulness; marked fluctuation." Paracentesis permitted the removal of 20,200 c.c. of yellow fluid, negative on examination. (Notes mislaid and exact details impossible to give now.)

Examination of the abdomen after removal of the fluid negative.

Patient was referred to the gynæcological clinic, where the examinations were likewise negative. I advised laparatomy, but on account of the negative examinations—two other gynæcologists had previously examined the patient and advised nothing but morphine tablets—this was declined.

The patient was lost sight of until May 4, 1906, when she returned in about the same condition as before. She said the fluid had slowly returned. She avoided salt and took a dose of Epsom salts in strong solution every other day. May 4 I again tapped, after a vain effort to get the patient to undergo a laparotomy. After removing a little more than 20 litres it was possible to feel the thick edge of the liver again, but as a new finding there was a large irregular hard movable mass just above the pubes.

After considerable urging the patient entered the gynæcologic clinic July 5, 1906, where laparotomy was performed (Dr. Peterson) July 6. "After opening the abdomen in the median line and evacuating a large amount of fluid, the liver was examined. It was small but soft. Gall-bladder empty. The surface of the spleen was rough (apparently fibroid patches, from the description.—G. D.). There was an irregular tumor the size of three fists occupying the pelvic cavity, fibroid of ovary. There were numerous adhesions between the bladder, peritoneum and fundus of the uterus." The tumor was removed, with the right ovary and both tubes. The patient made a good recovery, and has remained well.

There is a remarkable freedom from serious accidents in abdominal paracentesis, and this is doubtless one of the chief reasons for the persistence of a practice in many respects contrary to the present tendency in medicine and more especially in surgery. No doubt septic infection is lessened by the alteration of the serosa in many of the cases in which tapping is performed. But even with the greatest care, after the most complete examination, with every effort to choose a safe place to puncture, and with a cautious "palpatory" insertion of the trocar, accidents may follow, as in the following case:

Cirrhosis of the liver with ascites; tuberculosis of the peritoneum; rupture of a tuberculous focus in paracentesis; local infection of the peritoneum.

J. T. M., æt. forty, electrotyper, admitted to the hospital September 24, 1902, on account of large abdomen. Family history negative. Patient has drunk beer and whiskey regularly and for three years to excess, taking a pint of whiskey a day, besides occasional drinks of beer and gin. The present illness began four months before admission, with pain after eating, and frequent vomiting, at various times of day.

From the full notes on admission I take the most important data: "Emaciated. Enlarged venules on face, forehead, on abdomen between ensiform and navel, and on back above level of crest of ilium. Veins on front of thorax and abdomen enlarged and anastomosing. No hæmorrhoids. Œdema from feet up to top of sacrum. Thoracic organs pushed up. Apex beat in fourth and fifth interspaces, just inside nipple line. Soft systolic murmur over apex, transmitted to left axilla and to right. Radial pulse small, hard, regular. Abdomen distended, 38¼ inches around at level of umbilicus; bulges but little at sides; rises steeply with rounded top. Skin glossy over it. Movable dulness; marked fluctuation; testes negative.

"Blood: Red cells, 4,200,000; leucocytes, 5,100; Tallqvist, 90.

"Urine: No albumin, sugar, or diazo reaction. No casts."

The patient had been tapped before coming to the hospital and a few c.c. of bloody fluid obtained.

On passing a small trocar and canula September 26, in Monro's point, though the canula felt free, nothing came at first. Then a

few drops of opaque yellow fluid appeared, followed by turbid yellow fluid, specific gravity 1012. Two or three times the flow stopped again without any apparent reason. In all 7,700 c.c. were removed, having a specific gravity of 1013. Cells were chiefly small lymphocytes. No bacteria. A guinea pig inoculated with the sediment died in a short time, so the result could not be utilized. The venules on the abdominal wall became less marked. After the tapping the edge of the liver could be felt two inches below the ensiform in the middle line with a rather sharp edge and slightly uneven surface. The upper border on the sixth rib in the nipple line.

The fluid rapidly reappeared, and as it did the venules again became enlarged.

The diagnosis was cirrhosis of the liver, associated probably with tuberculosis. I endeavored to get the patient to consent to a laparotomy with a view to a Talma operation if possible, but without success. After six weeks, in which internal bleeding hæmorrhoids developed and albuminuria came on with hyaline and granular casts, the patient became unconscious during the night and died in this condition on the next day. There was slight fever, 99.5° to 102.5° for the first three weeks; later the temperature became subnormal, reaching 94° or less before death.

The autopsy revealed cirrhosis of an unusual type, periportal and central, with the portal much more advanced in the left lobe. In the right lobe central dilatation was marked; there was hyperplasia of bile ducts in all parts. There was hypertrophy of the heart, with slight fibroid change; slight arteriosclerosis. There was also acute miliary tuberculosis of all organs, rather sparsely disseminated in the lungs, liver, spleen, kidneys, lymph glands. In the peritoneum these tubercles were thicker and for the most part older. The oldest were in the omentum, where there were masses of confluent tubercles, many very large, with large areas of caseation. No evidence of puncture could be found in the omentum, but over an area of several inches in diameter about the site of the puncture through the wall, the visceral peritoneum was thickly set with yellow tubercles looking like a direct inoculation. I have no doubt the trocar broke one of the caseous masses, and that some of the contents that did not escape through the canula, produced the local infection.

Of course this played no distinct part in the course of the disease,

and not much could have been gained by any operation at the time the patient was first seen, but the lesson in regard to the danger of a trocar in the abdomen is obvious.

The subject of tuberculosis of the peritoneum is too large to consider *in extenso* now, but I shall give the brief notes of a case that shows, I think, the value of laparotomy in a variety of that disease, denied by many prominent surgeons.

Tuberculosis of the peritoneum with acute onset resembling typhoid fever. Laparotomy within four weeks of onset. Improvement. M. S., domestic, æt. 22. Was taken ill June 7, 1900, and when seen on the eighteenth gave a history and presented symptoms of typhoid fever, with unusual distention of the abdomen. However, the Widal rest was negative (leucocytes, 4,583), and on June 30 it was possible to detect free fluid, with a feeling of resistance to the right of the umbilicus.

July 2, Dr. C. G. Darling operated and found, as suspected, tuberculosis, with matted adhesions of the omentum. The patient made a good recovery from the operation, but later developed other masses in the abdomen. She is still alive, however, and has been able to earn her own living most of the time since the operation.

The next case I shall cite is one of the most instructive I have seen, from any point of view, and I need not comment at length upon it. Although in most cases of latent cancer masked by ascites a radial operation would be of little curative value, it could prevent much useless trouble and in some cases I believe that actual good could be done.

Cancer of the stomach with ascites as the first positive symptom. Ascites supposed, by exclusion, to be cardiac, after paracentesis. Diagnosis of malignant disease made a year later, near death, by rectal examination. P. N., aged sixty-six, lumberman, seen in consultation with Dr. J. W. Long, of Bryan, Ohio. Previous health good. Habits, including eating, good.

About one year ago patient began to complain of pain in the right side near the ribs. The pain was difficult to describe, but the patient thinks it was never colicky. He thinks he was never jaundiced, but others, including the doctor, thought there was a trace of jaundice for one or two days in the beginning. The abdomen gradually became larger, and was tapped. In September,

1901, consulted a prominent eastern consultant, whose letter is given below in abstract. A third and last tapping was made ten weeks before I saw the patient, and about 3 quarts of fluid removed. At this time the fluid was redder, as if containing blood, and coagulated rapidly after its withdrawal. Two weeks ago the patient took a rather long drive over rough roads. On returning he complained of pain in the abdomen, and since then has been much weaker than before. For the last few days there has been slight œdema of the face, hands and feet. There has been moderate constipation. Occasionally, lately, the patient has complained of sick stomach. He has emaciated considerably. The urine has always been negative.

June 20, 1902. The patient is now in bed. He complains of general weakness; there is marked emaciation; the face is thin but said to be somewhat fuller than a short time ago; the skin is dark, but not jaundiced, there are no areas of pigmentation. Appearance is cachectic.

The heart dulness is not enlarged, the sounds are very weak but regular, pulse small, tension low. Examination of lungs negative. The liver dulness is high but apparently not enlarged, the spleen cannot be felt.

At my suggestion the patient had been given an enema containing turpentine and castor oil. This caused the discharge of some flatus but I found the abdomen distended about three inches above the level of the ribs, the enlargement being greater below the navel. At times two oval prominences, about two by three inches in diameter, appear above the navel, raising up the abdominal wall. They are hard, elastic, disappear in a short time, and others form in the same position with or without manipulation of the abdominal wall. On disappearing no thickenings can be felt in the regions occupied by the tumors. There is no visible peristaltic motion in other parts of the abdomen, either spontaneously or as a result of irritation. Percussion of the abdomen gives tympany all over except about an inch above the pubes. There is no change of note with change of position in the sides, but the dulness rises in front about half an inch. This dulness became less, but did not entirely disappear, after passing urine. On palpation, the abdominal wall is moderately soft, but the distention interferes with deep manipulation.

Some nodules can be distinctly felt in the left nipple line above the level of the navel and in the right side below the position of the gall-bladder.

The rectum is empty; several small, hard, smooth nodules, about one to one and a half cm. long, can be felt behind the rectum. A rectal tube was passed up to the sigmoid, but no gas could be expelled.

The diagnosis lay between malignant disease of the peritoneum and tuberculosis. The cachexia, the absence of fever and the secondary nodules, especially those in the pelvis, suggested malignant disease. On the other hand, the early ascites, and difficulty of finding a primary focus made tuberculosis probable, and the tympanitic distention preventing deep palpation made it possible to exclude more characteristic tuberculous alterations. I questioned the patient especially with reference to stomach symptoms, on account of the great probability of the stomach being the primary seat. But both the patient and his physician, as well as the patient's relatives, all agreed that the stomach had not shown any symptoms until a very recent time. The urinary and genital tracts gave no evidences of primary alteration. The course of the disease did not warrant the diagnosis of cancer of the pancreas. The occasional occurrence of cancer of the gall-bladder with early secondaries in the peritoneum, and without local signs, made me think that the primary seat might have been there. The patient's weakness was such as to negative a test meal, or even diagnostic lavage. His condition was obviously hopeless, and it did not seem likely that the exact diagnosis would affect the treatment.

The patient died June 27; an autopsy was made thirty-six hours after death. There was a hard thick infiltration involving the lesser curvature and about half of the circumference of the stomach about that part. The mucous membrane of the stomach did not show any obvious ulceration in any part. The pyloric extremity of the stomach was not affected. There was also some thickening of the appendix, with nodules in the peritoneum in various parts. There were many adhesions between the intestines and other viscera. The pancreas and gall-bladder appeared normal, but there were so many adhesions about the pancreas and gall-ducts that a satisfactory examination was not possible. The liver seemed slightly congested and

enlarged, but contained no secondary nodules. The peritoneal cavity contained some thick yellow fluid about the consistency of molasses, apparently a mixture of embalming fluid and ascitic fluid.

Microscopic examination of the growth showed it to be scirrhus.

In the light of this later knowledge some extracts from the letter of the consultant are very interesting:

"I made a very careful examination of N., and, as you know, found hydroperitoneum in considerable degree. The only question is, to what is this symptom due. A hasty examination of the urine made at the time did not indicate any specific disease of the kidneys to suggest any relation, so far as they are concerned, and of course such a local accumulation with renal disease is of exceedingly rare occurrence, although I have seen several instances. One naturally expects in a localized hydroperitoneum that there is some local disease in the liver, or some local disturbance in the abdominal circulation, or disease of the peritoneum. There is nothing in the history to suggest any causation for cirrhosis of the liver; there is nothing to suggest portal thrombosis, nor is there anything aside from the gradual loss in weight and anæmia to suggest new growth in the abdomen. My first impression was that, considering the loss of weight, anæmia and age there was malignant disease somewhere in the abdominal cavity, with some secondary involvement of the peritoneum. After going very carefully into his history, and making more thorough physical examination of the heart, I am inclined to think that this interpretation is not correct, and that the accumulation of fluid is the result of disease of the circulatory apparatus. The history of the attack, shortness of breath, oppression in the chest, etc., two years ago, following rather severe exertion tends to confirm this interpretation. I fully realize the rarity of hydroperitoneum, as the result of disease of the circulatory apparatus, without general œdema; but again, I have seen a number of undoubted instances of this kind. The patient has certainly considerable disease of the myocardium. The character of the impulse, the heart sounds, quality of the pulse, all indicate this clearly. There is also considerable disease of the smaller arteries, and it may be that the vessels of the abdomen are more affected than the peripheral blood vessels. The arterial tension is not well maintained, because of the weakness in the heart muscle. It, therefore, seems

to me probable that it is disease of the heart muscle and the blood vessels which is accountable for the hydroperitoneum. I would advise comparatively dry diet, use of cardiac stimulants and tonics, with arsenic and small doses of iodide of potash. The digestion and bowels should be carefully regulated and the amount of exercise greatly restricted. I believe that it would be well to withdraw some of the fluid from the abdominal cavity, and probably when this is done, examination of the abdomen and microscopical examination of the fluid would either confirm the presence of, or exclude, disease in the abdomen."

But the autopsy shows that even the examinations suggested would in all probability have been negative. A cancer of the lesser curvature is usually impossible to palpate in its early stage. It also may cause no stagnation, so that the most important gross gastric evidences of cancer would probably have been absent. Perhaps a careful examination of the fasting stomach would have revealed evidences, such as bacteria, protozoa, blood or exudate cells, but the methods usually followed would almost certainly have been negative.

As for the peritoneal fluid, it would also be likely to be negative in the early stages, showing only a few leucocytes, and a large number of endothelial cells, indicating nothing more than irritation. In a recent case of ascites with cancer of the cardia, the fluid was of this negative character. A laparotomy was performed with the hope of doing gastro-enterostomy as a palliative measure. The whole stomach was involved, but there was no growth in the peritoneum. And yet in the case of N. it might have been possible early in the ascites to extirpate safely all the diseased tissue.

In conclusion, I would not wish to deny the propriety of abdominal paracentesis in all cases. For diagnosis it has a definite field, subject to the limitations I have stated. In treatment, it may at times be useful in inoperable cases, but before beginning this, one should be very sure that a more surgical procedure is out of the question.

PARALYSIS OF OCULOMOTOR NERVES IN DIABETES

BY PROFESSOR DIEULAFOY
Of the Paris Faculty of Medicine

Gentlemen: In the month of October, 1902, we had in our wards a man 73 years of age, of robust aspect, suffering from paralysis of the external oculomotor nerve of the left eye (the sixth pair). This paralysis was limited to the external rectus muscle, the other motor muscles of the eyeball being untouched, namely, those governed by the common oculomotor nerve (the third pair) and by the pathetic nerve (the fourth pair). The patient's left eye showed marked inward strabismus; he could move the eyeball in various directions, but all attempts at movements of abduction failed. At times a very faint trembling of the eyeball could be detected. All the movements of the right eye, on the other hand, were normal.

The patient complained of diplopia and vertigo, the diplopia being both binocular and monocular; he perceived two images, whether he used both eyes or one eye only, the other being held closed. Sometimes he even saw three images; looking at a house with a single entrance he had seen three doors, and, as was the case with his diplopia, this triplopia existed whether he used one eye or both, in fact the three images were even clearer when the right eye was closed.

His disordered vision was accompanied by a distressing condition of vertigo, particularly noticeable while walking, or on going up or down a staircase; the patient then closed his left eye instinctively, to avoid the annoyance of this sensation of vertigo.

The condition of the eyeball itself was perfectly normal, for a man of his age: the fundus and lense were sound, the pupils equal, the light reflexes and power of accommodation as they should be. There was neither disease nor malformation of the eye itself.

This paralysis of the sixth pair had appeared suddenly, though it had been preceded by a period of pain; for a week previously the patient had suffered from severe pain in the left temporal region, with periorbital radiations. This was not an ordinary headache,

but an attack of temporo-orbital neuralgia; the pain increased on pressure, was almost continuous, and interfered with sleep.

The night that preceded the attack of paralysis had been particularly troubled, and in the morning a neighbor of our patient exclaimed as he saw him: "What is the matter with you, your eye is all to one side!" The paralysis of the external oculomotor nerve was then already complete, with its classical signs, inward strabismus and diplopia; but what was less usual was the temporo-orbital neuralgia with triplopia.

Our first duty was to ascertain the cause of this paralysis of the sixth pair, since the prognosis and treatment depended altogether on the pathogenic diagnosis.

Among the various causes that may give rise to paralysis of the oculomotor nerves, syphilis and tabes take the lead, the latter being in the great majority of cases, merely an offshoot of syphilis.

The question of syphilitic paralysis of the oculomotor nerves has been carefully studied by Professor Fournier, from whom I quote the following: "Cerebral syphilis does not affect with equal frequency the three pairs of nerves that govern the movements of the eyes. Thus it rarely attacks the pathetic, or fourth pair. On the other hand it shows a certain predilection for the sixth pair, the external oculomotor nerves. But its great field of action is the third pair, the common motor nerves, which it attacks with far greater frequency than the other two. The ordinary ocular syphilitic paralysis is that of the third pair, and there is no exaggeration in saying that such a paralysis has a specific mark of the disease. Again, this paralysis of the third pair very commonly appears in a partial form; instead of the entire distribution of the nerve being affected, only certain branches lose their power. In such cases we find, either alone or combined, ptosis, outward strabismus, paralysis of the upper rectus, or mydriasis. Partial paralysis of the third pair is extremely common in cerebral syphilis, more common than in any other disorder, and on this account this symptom is of great diagnostic value."

But our patient was not a syphilitic; the most careful interrogatory failed to find any signs of this disorder, and we were consequently obliged to eliminate syphilis as the cause of his paralysis.

Next as regards locomotor ataxia,—you know that ocular paraly-

sis is not rare in tabes, and that it affects in order of frequency the third, sixth and fourth pairs, and that when it affects the third pair the paralysis is often only partial, dissociated. In time of appearance, ocular paralysis in tabes is either early, or late. Early ocular paralysis is usually less serious and less permanent than paralysis of confirmed tabes; it lasts only a few weeks, hardly a few months, and in some cases only a few days. It appears and disappears rapidly, and its character is to be monocular, partial, affecting only a single or a few terminal branches of the oculomotor nerves. According to the branch affected the patient manifests strabismus, diplopia, ptosis, etc., and relapse is not unusual.

Late tabetic ocular paralysis, occurring at the confirmed stage of the disease, has an entirely different evolution from that of the form just described; it follows a slower course, does not usually improve, and sometimes affects both eyes. It also differs from early paralysis in the nature of the lesion on which it depends, since early, transitory paralysis seems to be caused by peripheral neuritis, whereas the late form is due to lesions in the bulboprotuberantial nuclei.

But again, with our patient, we had to set aside tabes as the cause of his ocular paralysis, as he showed no signs whatever of the disorder. We were therefore obliged to look elsewhere for the explanation of his condition.

Many other disorders: general paralysis of the insane, tumors of the brain, cerebral tuberculosis, are capable of causing ocular paralysis; but these different causes had also to be set aside in the present instance.

There then remained a disease that will have to be taken seriously into account in the pathogenesis of ocular paralysis,—I refer to diabetes. Now our patient was a diabetic; he had marked polyuria and great thirst, drinking each day six to eight quarts of liquid and urinating in proportion.

An analysis made gave the following results:

Density	1030	
Quantity of sugar per liter	23	grams
Quantity of sugar per 24 hours	138	"
Urea per 24 hours	69	"
Phosphates per 24 hours	6.16	"
Albumen	0.	"

This case of diabetes was already a long-standing one; it appeared to have begun seven years previously with extreme thirst, and a few months later as much as 480 grams of sugar in the 24 hours had been detected in his case. Later on a number of diabetic complications had occurred: profuse epistaxis, gingivitis, and expulsive periostitis, with loss of several teeth.

I therefore made, in the case of our man, the diagnosis of diabetic paralysis of the sixth pair, and I told you that this paralysis would disappear in two or three months. I prescribed one gram of antipyrin per diem, and the use of the galvanic current. The patient was put on a mild regimen; sugar was forbidden, but some feculents were allowed, especially potatoes. The temporo-orbital pain lasted for a couple of weeks still; the symptoms of paralysis decreased steadily, and in three months time strabismus and diplopia had entirely disappeared. Since that time, that is, for three years, our patient has continued to have his marked degree of diabetes, which does not seem to trouble him much; but he has not shown any further signs whatever of paralysis of the oculomotor nerves.

On two other occasions I have seen cases of diabetic ocular paralysis, in the following circumstances:

Five years ago I met one day on the street a friend with a little opaque disc fixed to the brim of his hat and coming down in front of his right eye; this disc served to alleviate a very annoying condition of diplopia. I ascertained that he was suffering from paralysis of the sixth pair, that this paralysis had occurred suddenly, and that he was much affected by it. He related his gloomy fears to me; it seems that he dreaded the onset of cerebral trouble, and even spoke of the possibility of tabes or paresis. But I happened to know all about his health, that he had not had syphilis, but on the other hand was diabetic. I therefore made a diagnosis of diabetic ocular paralysis, reassured the patient, and told him that the symptom would disappear in a few months and he would not hear of it again.

For many years I have had under my care a gouty patient with diabetes, the quantity of sugar never exceeding 30–40 grams per diem. As this man was quietly sitting in the Avenue du Bois, one day five years ago, he noticed two men pass by him on horseback so

exactly alike that he was dumbfounded, having never seen such a remarkable resemblance before. What he noticed was in reality the effect of diplopia, for at that moment his double sight had begun, unknown to him, and there was only a single rider in front of him. A few moments later he perceived two ladies, also exactly alike, and each one leading the same pet dog. By that time, feeling quite bewildered, giddy, uncertain as to his sight and at a loss to understand what was happening, he took a cab, drove home and sent for me in all haste. I found him quite beside himself, dreading an attack of cerebral congestion, or of apoplexy. His left sixth nerve was paralyzed, and as I had long been aware of his diabetes, and knew on the other hand that there was no syphilis in the case, I reassured his fears, and told him that the paralysis was due entirely to his diabetes and would disappear in a few months. Since that time, that is, five years, his paralysis, which was cured in two months and a half, has not reappeared, though he is still diabetic, naturally.

These cases made a strong impression on me, so much so that in order to look into this question of ocular paralysis in diabetes more closely, I have consulted a number of the best-known eye-specialists on the point of their frequency, and wish here to thank them for the interesting data with which they have so obligingly furnished me.

By adding up the cases of paralysis of the oculomotor nerves in diabetes reported to me by these competent observers (upwards of twenty in number) I have obtained a total of 58 cases, namely: 35 cases of paralysis of the sixth pair, 12 cases of paralysis of the third pair, 5 cases of paralysis of the fourth pair, and 6 cases of external ophthalmoplegia,—without counting the cases observed by some other oculists who had failed to make notes of them.

In all the cases just mentioned nothing but diabetes could be suspected, all other causal factors being lacking, the patients being neither syphilitic, tabetic, nor hysterical, and suffering neither from paresis, nor cerebral tumor; they were only diabetic.

These details being understood, let us consider a few points connected with this ocular paralysis occurring in diabetes.

A first peculiarity is that paralysis of the sixth pair is much the most common form,—three times more frequent than paralysis

of the third pair; whereas in syphilis and tabes it is partial or entire paralysis of the third pair that is generally encountered. Whence the precept that when you find yourself in presence of a case of paralysis of the sixth pair, diabetes is the cause first to be thought of. This kind of paralysis may even attain the importance of a symptom betraying diabetes; a glycosuric patient may have neither increased thirst nor polyuria, in fact none of the usual signs of diabetes, when the paralysis of the sixth pair occurs. An analysis is then made and the glycosuria is discovered.

In this connection it is well to remark that paralysis of the oculomotor nerves in diabetes does not depend on the degree of the glycosuria; in one case the paralysis appears with a patient eliminating sugar by hundreds of grams a day, while other patients will manifest the same symptom with only 20 grams, 12 grams, or even 3 grams a day. In fact, the glycosuria has been known to be temporarily absent at the moment when the paralysis occurred. There is therefore no direct relation, and the point is important, between the degree of glycosuria and the appearance of oculomotor paralysis.

These attacks of paralysis are generally rather sudden, but their duration is not a long one. As a usual thing the symptoms begin to improve after a few weeks, the paralysis having quite disappeared in two or three months; on the other hand certain cases recover more rapidly still, while others last even longer.

Recurrence is not rare. Thus a diabetic patient who has already had an attack of paralysis of the sixth pair, from which he entirely recovered, can, a few months or years later, have another attack of paralysis of the sixth, third or fourth pair, affecting one eye, or both.

Among the 58 cases above referred to, in five instances the paralysis affected all of the oculomotor muscles, in the form of unilateral or bilateral external ophthalmoplegia. In a case reported by Blanc the ophthalmoplegia affected both eyes and was followed by paresis of both hypoglossal nerves and of the inferior facial nerves. In Saurineau's case the ophthalmoplegia was progressive and unilateral, and recovery took place within a few months. In another case of the same writer his diabetic patient had first an attack of paralysis of the left third pair, and eighteen

months later, progressive ophthalmoplegia of the right side lasting eight months.

This goes to show that these cases of ocular paralysis in diabetes are not always light and transitory. In a few instances they are tenacious and of an extending nature, and affect in the form of progressive ophthalmoplegia the oculomotor nerves of one or both eyes.

There are even cases, extremely rare it is true, in which the ocular paralysis of diabetes may be the forerunners of the most serious conditions; thus an attack of paralysis, apparently harmless, may before long turn into ophthalmoplegia, and end in fatal polioencephalitis. Such a case was Koenig's, of which the details are as follows:

"One of my cases went through a rapid evolution, and the chronic upper polioencephalitis ended in a bulbar phase. The external ophthalmoplegia began by left conjugate paralysis, which was followed by loss of associate movement on the right side. Ptosis appeared on each side alternately, the other movements of the eyes soon became affected, and the lower lids relaxed. Two months later the paralysis affected the upper and lower facial nerves. This phase preceded the appearance of bulbar symptoms (polyria, facial anesthesia, glossoplegia), in the course of which the patient succumbed. This is the picture of true progressive nuclear ophthalmoplegia. With this diabetic patient the analysis had furnished the following results: Sugar, 65 grams per liter, with four liters per 24 hours."

Such are, as regards their evolution, the different forms of ocular paralysis in diabetes. You have seen that in the great majority of cases,—nine times out of ten,—these attacks of paralysis are extremely mild; they are not an indication of danger, and get well in a few months without leaving any trouble behind them. It is therefore important to be well acquainted with them, in order to reassure the diabetic patient who may be seized with one. Such patients are liable to be very much upset and alarmed by the suddenness of such an occurrence, and by the nature of the disturbance that has come on them; they apprehend cerebral trouble and think that their attack is a commencement of apoplexy. Their fears can be assuaged; nevertheless the exceptions must also be taken into account, and you must not forget that any attack of ocular paralysis,

no matter how mild, may with diabetic patients end in external ophthalmoplegia of long duration and even in very serious bulbar symptoms.

I now wish to refer again to one of the signs presented by our first patient. I told you that this diabetic patient had suffered during the week preceding his attack of paralysis of the sixth pair, from violent and continuous temporo-orbital neuralgia. The pain was strictly localized, did not extend to the rest of the face, was not increased by pressure, continued day and night without respite, and did not cease until two weeks after the appearance of the paralysis. This was not an ordinary headache, but appeared to be a form of neuralgia depending on the same cause as the paralysis,—in a word, my impression was that our patient was suffering from an attack of *painful paralysis* of the sixth pair. Although the element pain, combined with paralysis of the oculomotor nerves, does not seem, to my knowledge, to have specially attracted anyone's attention, nevertheless I have noted this symptom casually mentioned in connection with several of this series of cases.

Thus in one of M. de Lapersonne's diabetic cases the paralysis of the sixth pair was accompanied by temporal neuralgia. In one of Galezowski's diabetic cases the paralysis of the third pair was associated with such violent pain that he had at one time thought the case to be one of ophthalmic zona. In one of Koenig's diabetic cases the paralysis of the sixth pair had been preceded and accompanied by pain about the right eye. With Nattan-Larrier's diabetic patient the paralysis of the sixth pair had been preceded by violent temporo-orbital neuralgia, which continued during the entire duration of the paralysis and only disappeared when the latter was cured. With a diabetic case of Buge's the paralysis of the fourth pair was accompanied by pain in both temples. In one of Saurineau's cases the paralysis of the left third pair was accompanied by slight pain in the left side of the head. In one of Deléage's cases the paralysis of the sixth pair was accompanied by very severe pain about the orbit and in the left temporal region.

This makes you see a fair number of diabetic patients with whom an attack of paralysis of an oculomotor nerve has been preceded, accompanied or followed by neuralgic pain in the temporal region, with or without periorbital radiations on the same side as

the paralysis. This is not a mere coincidence; the two symptoms, pain and paralysis are associated.

From a clinical standpoint this fact seems to me to be of such importance that when a diabetic patient complains of severe pain in the temporal region and about the orbit, we must be on the lookout for the possible appearance of paralysis of an oculomotor nerve.

.Perhaps the explanation of this association of neuralgia and paralysis may be found in the following fact brought forward by Bonnier. Deiters' nucleus is in direct communication with the oculomotor centers; and this nucleus, as has been proved by Probst, is connected with the descending, sensitive root of the fifth pair. These connections may perhaps explain the way in which a single cause acting on a given region may produce both paralysis and neuralgia. However this may be, and whatever the correct theory may be, the patent fact, on which I am anxious to lay stress, is that with diabetic patients, paralysis of the oculomotor nerves is quite often preceded and accompanied by severe neuralgia of the temporal region and about the orbit.

After this examination of the question of paralysis of the oculomotor nerves in diabetic patients, let us discuss a little the pathogenesis of this paralysis. How can diabetes paralyze the nerves of the eye, and how much of this action is due to the glycosuria,—such are the points yet to be solved.

It is generally admitted that diabetes can produce toxic neuritis, and to this cause has been ascribed the diabetic-sciatic neuralgia, the frequency of which in this disease is well known. To this form of neuritis also have been attributed cases in which paralysis and muscular atrophy occur together: atrophic paralysis of the muscles governed by the ulnar nerve (Ziemssen), atrophic paralysis limited to the domain of the external peroneal nerve (Bernard and Ferré). Are we then to attribute paralysis of the motor nerves of the eye also to diabetic neuritis? But if diabetes (by some badly understood mechanism) is really capable of intoxicating the motor nerves of the system, how does it come to spare the motor nerves of the limbs, in order to reserve its baneful effect for the motor nerves of the eyes? For, so far as I am concerned, I am not aware of a single instance in which diabetic paralysis has affected the nerves of the limbs, whereas I have been able to gather together in this paper 58

cases of paralysis of the motor nerves of the eyes. There is here such flagrant discordance that the hypothesis of diabetic neuritis as a cause for this paralysis will I think have to be abandoned. Besides, we should have to allow a high toxic power for diabetic sugar, when we see ocular paralysis occur with patients having only a few grams in their urine. Now no one has ever demonstrated any such toxic power. For all of these reasons I cannot accept the idea that these cases of paralysis are due to neuritis depending on some poison derived from the sugar. In this question of pathogenesis the sugar is a witness only, and not the cause of the disturbance.

On the other hand it is impossible not to be struck by the quite peculiar localization of these attacks of diabetic paralysis. Why is it that they affect the oculomotor nerves almost exclusively? When we consider this point our attention is necessarily drawn to the region of the nerve-centers comprising the fourth ventricle and the origin of the oculomotor nerves. And then Claude Bernard's memorable experiment comes before the mind, and we remember that the region which when pricked gives rise to glycosuria is very close to the nuclei that are the origin of the nerves which are paralyzed with diabetic patients. This connection of ideas cannot well be avoided, and, hypothesis for hypothesis, the latter seems fully as good as the former. In the immense majority of cases the bulbar symptom-complexus is reduced to its simplest expression, namely: glycosuria (marked or slight), with paralysis of some nerve, usually one of the sixth pair. In other circumstances the bulbar symptom-complexus is more extensive, and in addition to more or less marked glycosuria we have paralysis of several oculomotor nerves and even external ophthalmoplegia, unilateral or bilateral. Finally, the bulbar symptom-complexus may acquire exceptional gravity, as in Koenig's case mentioned above, in which fatal, progressive nuclear ophthalmoplegia was added to the glycosuria.

As for explaining the origin of this morbid association, it cannot yet be done. Even supposing that a single cause can produce both glycosuria and oculomotor paralysis, it is difficult to grasp why this single cause should have such a persistent action on the glycosuria, and so temporary a one on the paralysis.

Note that the forms of ocular paralysis which have been examined do not occur in insipid diabetes, but in diabetes mellitus.

THE DETECTION OF THE OVA OF INTESTINAL PARASITES IN THE FÆCES

BY MAURICE LETULLE, M.D.
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In clinical medicine the usefulness of the microscope—no longer disputed by anybody—is found to be of striking value when the physician finally decides to examine the fæces of his patients as a routine matter. When the existence of some disorder caused or perpetuated by intestinal worms is suspected, histological examination of the stools is a necessity.

The worm-diseases of man, which for too long a period were relegated among the legends of ancient pathology, have fortunately regained among us in recent times the favor of the medical public. The increasing frequency of the relations between our metropolis and the colonies has obliged us to take up the study of exotic pathology, in which the parasitic disorders, and in particular those of the intestine, take such a prominent part. In Paris our hospital wards constantly contain patients from the colonies who import into our midst, besides malaria and amœbic dysentery, the greatest variety of tropical disorders, among which may be especially mentioned anchylostomiasis and bilharziosis.

Methodical examination of the fæces, clinical copsology, should be a matter of principle in a number of circumstances where the physician's attention is directed to the intestine for one reason or another,—persistent anæmia, stubborn diarrhœa, appendicitis, etc. But for the present we will only consider the microscopical treatment of the fæces in looking for the ova of the parasites of the intestine.

The necessary outfit of instruments is very simple: a good microscope, an ordinary eye-piece (No. 6, stiassnic, for instance), two lenses, an average one (No. 2, stiassnic), and a stronger one (No. 8, stiassnic), a few clean slides and cover-pieces, a glass pipette, a platinum thread for the reagents, a weak solution of formol (1 or 2 per cent), a concentrated solution of acetate of potash—this is

all that is required, and the articles can serve equally well in many other researches in clinical microscopy.

The preparatory technique is as little complicated as possible, though varying somewhat according to whether the fæces placed at our disposal are fluid or solid.

When the fæces are liquid they can, if desired, be treated by a centrifugal apparatus; but there is danger with this treatment of damaging the ova that are projected violently against the bottom of the tube (bilharziosis, uncinariosis). As a general thing it is preferable to make a greater number of slides, after decanting the liquid, and to look for the suspected ova on a larger number of preparations. With liquid fæces the material may be taken with a pipette, a drop deposited on each slide and immediately covered with a cover-glass. Care should be taken that no air-bubbles are included in the fæcal drop between the two slides.

An initial precaution, very advantageous from many points of view, consists in diluting the fæces with a small amount of the 1 to 2 per cent. formol solution. This both destroys the odor of the fæces and preserves the ova in the intestinal fluid. But in some instances, as, for example, when intestinal bilharziosis is suspected, there is a particular interest in being able to observe the vitality, that is to say the contractile power of the ova of the incriminated parasites. It is therefore prudent always to begin by preparing a few slides without the addition of any foreign liquid, except for a little sterilized water, should the fæces be too concentrated.

As soon as the extemporaneous preparation has been made, the microscopical research can begin at once.

If it is desired to have a permanent preparation of the fæces in which ova have been detected, several methods, easy and reliable, may be mentioned. The best one consists in exposing, for one or two minutes, to vapors of osmic acid (a 1 per cent. solution of water in a wide-mouthed vial) the fæcal drop hanging from the slide turned upside down. In this way the ova are quickly preserved. The drop prepared after this process can be set either with pure glycerine, in glycerinized water (1-30), or in acetate of potash, cedar balsam; or, better, cedar oil, may be used, though this technique requires an absolutely anhydrous preparation. In any event it is indispensable to harden the ova thoroughly before the

final process, as glycerine, when brought into contact with living ova, is capable of modifying them, and even of rendering them entirely unrecognizable by inhibition.

When the fæces are solid, they must be diluted in the formol solution,—after which the technique is the same as above.

Microscopical Examination of an Ovum.—When an ovum is perceived in the field of sight with an average enlargement, how shall we know to what category it belongs?

We must first be sure that it is indeed an ovum, and to that end we must at least be familiar with drawings of the different ova that can be found in the human fæces. Six chief varieties (not to speak of the tænias) may be met with in stools. These are, in the order of their frequency:

1. Trichocephalus.
2. Oxyuris vermicularis.
3. Anchylostomum duodenale.
4. Bothriocephalus.
5. Ascaris lumbricoides.
6. Bilharzia hæmatobium.

Whenever an ovum appears on the slide it is well always to conduct the examination in accordance with a given method, in order to perceive its different distinctive characteristics. The following order will, I believe, be found satisfactory:

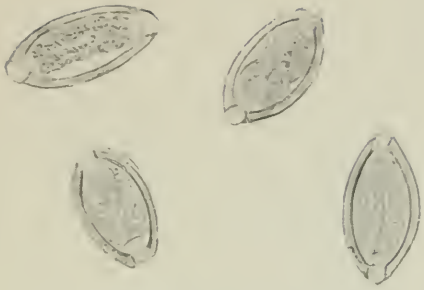
Examine, first, its shape; second, color; third, transparency; fourth, micrometric dimensions; fifth, structure (*a,* of the shell; *b,* of the contents); sixth, artificial changes produced by the reagents applied; seventh, signs of vitality (contractile power) appreciable through the microscope; eight, relative abundance of the ova in the different preparations examined.

The following are the general characteristics of the six kinds of ova most usually found:

1. *Trichocephalus.* This is the easiest one to recognize, on account of its lemon shape and its brownish-yellow color (Fig. 1.); at either extremity of the ovum there is a depression surmounted by a sort of pale, translucent drop, a peculiarity in a way specific.

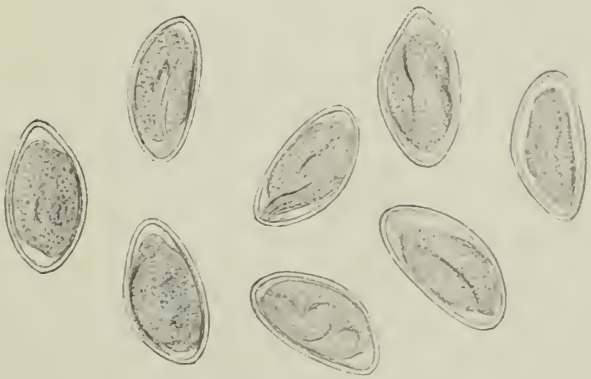
The usual dimensions given are 51 to 53 microns in length, by 21 to 23 microns in breadth. Those that I have observed have been somewhat greater: 57–61, by 25 or 26.

Fig. 1.



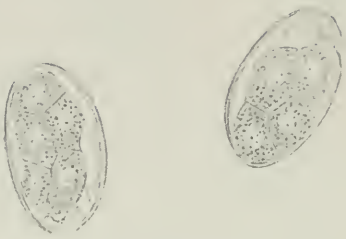
Ova of Trichocephalus dispar.

Fig. 2.



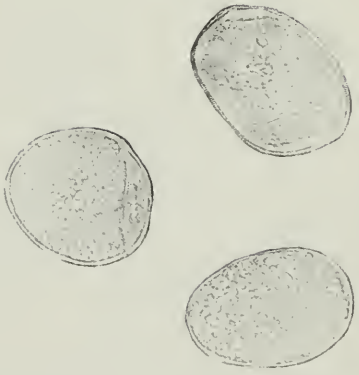
Ova of Oxyuris vermicularis.

Fig. 3.



Ova of Anchylostomum duodenale.

Fig. 3*b*.

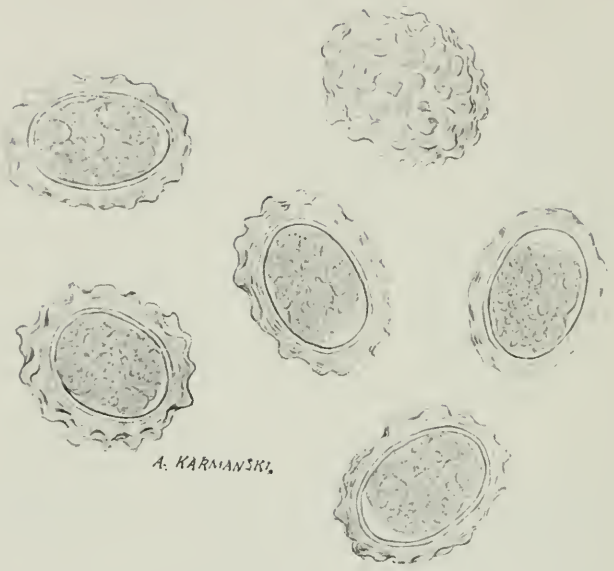


Ova of Anchylostomum duodenale altered by glycerinized water.

Fig. 4.

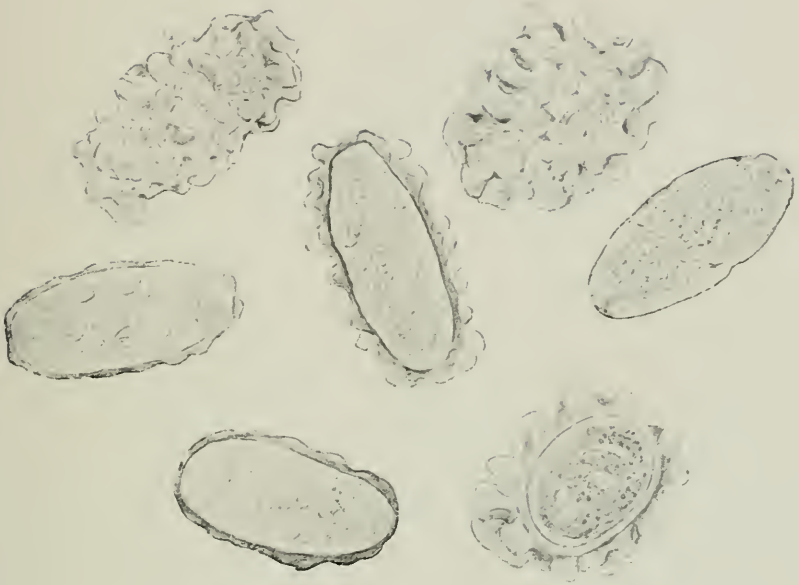
Ova of Bothriocephalus latus.

FIG. 5.



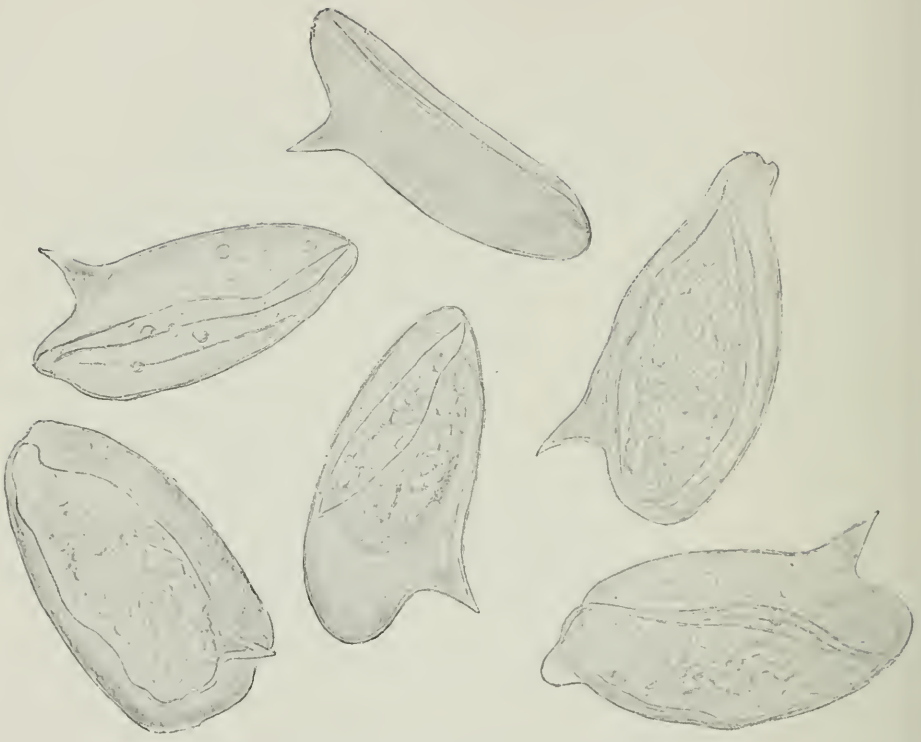
Ova of Ascaris lumbricoides (early stage of development)

Fig. 56.



Ova of Ascaris lumbricoides (later stage of development).

Fig. 6.



Ova of Bilharzia hæmatobium (all the ova are empty).

2. *Oxyuris vermicularis.* This parasite, formerly so common among Paris children, is becoming more and more infrequent. The ova (Fig. 2) are quite easy to detect, owing to the asymmetrical irregularity of their shape; the embryo is often readily perceptible within the shell.

The usual dimensions given are 50 to 54 microns in length by 20–27 microns in width. My own figures are somewhat different, as in a great many preparations I have found the following: 59–61 microns, by 27–33 microns.

3. *Anchylostomum duodenale.* This parasite is to be found not only in mining regions in France, where it causes the anæmia that affects miners, but I have had this year two cases in my hospital wards: one in a Congo negro, who was also suffering from bilharziosis, and the other in an anæmic girl who had arrived from Brazil. The ova (Fig. 3 and 3b) are most characteristic; their white color and shiny, silvery brightness at once attract the eye.

The usual measurements, too small, are 52–58 microns in length by 32–35 in width. My own figures, considerably greater, perhaps because I measured the ova alive, in fresh fæces not treated by any reagent, are 62–69 microns by 42–47 microns. It is common to detect in the inside of the shiny, pearly shell the constituent elements of the embryo in the shape of large, nucleated cellular elements (Fig. 3). Glycerine soon alters the embryo (Fig. 3*b*).

4. *Bothriocephalus.* The ova of this parasite (of which the drawings published in the various books are much too artificial) can be recognized by their size, their irregularly rounded shape (Fig. 4), and lastly by their line of opening, which is far from forming the valve usually represented in the drawings, where, as pictured by the illustrators, it is as elegant as it is regular. The dimensions of the ova vary much according as to whether a small or a large ovum is selected. The usual figures, 68–70 microns by 44–45, do not give an accurate idea of the differences with which one meets. Thus with small ova I have found 57–64 by 43–45; whereas with large ones, 71, 76 and even 78½ microns by 47, 52 and even 59½.

5. *Ascaris lumbricoides.* The same remarks are applicable to the ova of this parasite. Fortunately this ovum is so easy to detect by its brownish-yellow color and the somewhat mulberry appear-

ance of its envelope (Fig. 5) that no mistake is possible; within the thick and rough shell the embryo, already formed, can often be recognized (Fig. 5*b*).

Here once more the dimensions are very variable, since in the case of a child I found ova (Fig. 5) almost spherical and measuring 59 and 54 on an average, whereas with several adults, and particularly in the case of a Congo negro, ova 95 and even 104 microns in length by 46 and 57 in width were the rule (Fig. 5*b*). The usual figures that only vary between 65–75 in length and 40–58 in breadth are therefore misleading.

6. *Bilharzia hæmatobium.* These are the largest of the six classes of ova that we have mentioned. They are so characteristic that a mere drawing is enough for their description by itself. Very large, oviform, but provided almost all with a lateral spur, these ova appear in the fæces almost always broken open and empty.

For my part I have never found a living ovum in fæces, whereas in urine I have found as many as I wished. The shell is dull, yellowish and thick; the spur, often long (23.8 microns on an average), is generally intact.

The dimensions usually given are 135–160 microns in length by 55–66 microns in width. My own figures, taken from a single case, were not so great: 126 in length on an average, and 52 in width. The live ovum is naturally larger than the broken and empty one.

In short, the microscopical study of the ova to be found in fæces is indispensable for the physician. The technique is easy, and the diagnosis certain. And the advantages to be derived from this method are immense as compared with the slight inconveniences of a means that is simple and within the reach of every one.

THE CLINICAL EXAMINATION OF THE CEREBROSPINAL FLUID

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With the extension of the use of lumbar puncture as a means in diagnosis, has come the necessity for careful determinations on the cerebrospinal fluid. Many of its qualities have been well studied, but of these part, as the freezing-point, the toxicity, and the electrical conductivity, have no essential value for the clinician, and part are of more interest than importance, as, for example, glucose-content during a diabetes, and the appearance of urea during uræmia. Indeed, the characters of the fluid that, by reason of their use, demand determination are but few,—the gross appearance, the bacteriology, the cell-content and cell-type, the protein content, and the general intraspinal and intracranial pressure as expressed by that under which the fluid is held in the subarachnoid space. Despite the worth of all of these, as repeatedly proven, the examination of a specimen, as ordinarily performed, includes notes only on the first two, with guesses, more or less careful, as to pressure and cells present. Under such cursory investigation many pathological fluids appear normal and their significance is passed over, as in the case of those clear fluids of tuberculous meningitis, in which no bacilli have been found, and of those, which seem equally innocent, from patients with paresis. This happens because there is at present no defined routine technic (apart from the bacteriological) for the examination of specimens of cerebrospinal fluid. To discuss a technic is the purpose of this paper.

In connection with the operation of lumbar puncture itself,—now excellently understood,—a caution or two may be given in passing. Death has often followed the withdrawal of much fluid. Usually this has occurred when the pressure in the subarachnoid space has been high and its rapid change in draining of the fluid has worked injury to vessels already sclerosed or to parts encroached on by a neoplasm. It is but natural that the operator, seeing the

fluid spurt vigorously from the canula, should, arguing from a therapeutic basis, allow more to escape, than when the fluid drips. Such a course is to be warned against as is also that of "draining" to the full the meninges of a patient with meningitis. In a well-grown child of 12 years with tuberculous meningitis, recently under my observation, an immediate collapse followed the withdrawal of 30 cc. of cerebrospinal fluid. For the purpose of examination not more than 6 cc. are needed. In the healthy, the removal of 10 cc. is not infrequently followed, as Nissl showed, by nausea and headache persistent for a day or so. These symptoms are especially likely to occur if the patient is not kept in bed for a few hours following the puncture.

Not less vital is the proper control of the needle during its insertion into the vertebral canal. It is quite possible with an 8 cm. needle, as may be readily shown on the cadaver, to thrust into the peritoneal cavity or between the bodies of the vertebræ, and by this latter path to pierce even the vena cava. For such reasons the exact orientation of the needle point should always be in mind. Aspiration to start the flow of fluid is not permissible, and indeed should not be necessary if the bore of the needle stay clear. To ensure this a wire plunger, of the exact length of the bore, is kept in the needle during its insertion and removed only when the operator feels that the subarachnoid space has been entered. Thus "dry taps" are avoided. Indeed, a "dry tap" is not conceivable except under the condition of complete absence of fluid about the cauda equina in the region of the puncture,—a condition hypothetically possible in certain pathological states, but not encountered clinically.

For exact pressure determinations elaborate apparatus is required, but for ordinary bedside purposes the device already described elsewhere, in use now for some years at the Johns Hopkins Hospital, suffices. As soon as the subarachnoid space has been entered, as shown by the appearance of fluid, a thick-walled glass tube of fine bore (preferably of about 1½ mm. diameter), held vertically, is joined to the needle by a short, rubber connection, and the pressure is measured in terms of the height of the column of fluid maintained in the tube. The capillary error, the variations in specific gravity of the fluid, and the amount of it leaving the subarachnoid space to form the column (at most 1 to 2 cc. when the

diameter is as cited) may all for practical purposes be neglected. Krönig with careful experiment and special apparatus found the normal pressure to be equal to 120 to 180 mm. of distilled water, but under ordinary bedside conditions with this improvised manometer the range is much wider, 50 to 300 mm. of this fluid itself. Crying or coughing on the patient's part will increase this 50 mm. or more, temporarily; and that the position of the body influences pressure is shown by the increase in height of the column when the patient,—who should always be lying horizontally on his side,—raises the head. The column further shows a fluctuation with the pulse and the respiration, except in the extremely æsthenic, or when the pressure connection between the subarachnoid space and the column of fluid in the tube is broken. From all this it follows that the measurement should be taken when the patient is, so far as possible, relaxed, and that gross differences in pressure are alone of much significance.

As has been said, the normal pressure range may be stated to be between 50 and 300 mm. of the fluid itself. A marked increase over this is the rule in hydrocephalus, in brain tumor, and in meningitis of bacterial origin, especially when acute. Often an increase accompanies uræmic coma. A column 600 to 700 mm. in height is not uncommon in any one of these conditions. Low pressures are observed in infants, in conditions giving low blood pressure, and when there has occurred a technical error.

With the pressure determined, one disconnects the manometer, and collects the cerebrospinal fluid as it drips or spurts from the hollow needle. One or two cc. may be taken under aseptic precautions for the bacteriologist, and 4 to 5 cc. more allowed to run directly into a clean centrifuge tube.

The determination of cell-quantity is now immediately in order. The method in vogue for this is that of Widal, Sicard and Ravaut, described in 1900. A given quantity of fluid is centrifugalized for a certain time at a known rate of speed, smears are prepared of the sediment, and, according to the number of cells found per oil immersion field, one judges of the presence or absence of a "spinal leukocytosis." The errors as well as the disadvantages of this procedure are obvious and great, vitiating the bulk of observations made by it. Much better than this is a count of cells with the Thoma-Zeiss appa-

ratus, as Fuchs and Rosenthal demonstrated in 1904. But their diluting solution of acetic acid and methyl violet destroys erythrocytes, and, in consequence, when a specimen has been contaminated with blood, destroys the index whereby one might tell to what extent the white cells present are due to the contamination, and to what extent they are intrinsic to the fluid. Instead, 4 parts of a saturated aqueous solution of methyl violet (5 B.) may be added to 996 parts of the cerebrospinal fluid in the ordinary 1 to 200 mixing pipette, as proposed by the author. Thus the leukocytes are stained and the erythrocytes present retain their normal appearance, unless they have previously been laked, as occurs in some specimens. In the absence of this laking, an absence attested by intact red cells and the lack of diffused hæmaglobin (proven by the colorless supernatant fluid obtained on centrifugalization of another part of the specimen), these red cells may be counted as well as the white, and allowances made for the number of the latter due to blood contamination, on the basis of the relation between the two in the blood. Say, for instance, a case of typhoid fever has "meningismus," and on lumbar puncture a small vessel is wounded and a quite bloody fluid, apparently hopeless as cytological evidence, is obtained. The red cells in the specimen have not been laked, and there are 80,000 of them and 102 white cells per c.mm., with, in the blood itself, 4,000,000 r.b.c. and 5,000 w.b.c per c.mm., or a proportion of 800 to 1. Calculating on this ratio, of the 102 white cells present per c.mm. in the cerebrospinal fluid, 100 are from the contaminating blood. Thus but 2 w.b.c per c.mm., a normal number, were originally present in the fluid, and one has the strongest presumptive evidence that no meningitis is present. Had the white cells in the fluid numbered 1002 per c.mm., instead of 102, one would have, making certain that few or no red corpuscles had been laked, equally strong evidence toward meningeal trouble. Thus it often proves possible, in presence of even a considerable trace of blood, to tell a normal fluid from a pathological so far as regards cell-number.

Here must be noted those cases in which the blood may be the expression of a previous intracranial or intraspinal hæmorrhage. Under such circumstances it becomes of the utmost diagnostic value, if one can be certain that it is not derived entirely from an injury caused by the puncture. Blood from the latter source

is frequently abundant in the first few drops of fluid and is not seen in that flowing later, while, furthermore, slight alterations in the position of the needle will often notably affect its quantity; whereas that derived from a previous hæmorrhage is more evenly distributed, and remains unchanged in amount despite gentle manipulation of the needle. Some have held that the red cells of a previous hæmorrhage, as contrasted with those entering during lumbar puncture, are always broken down, but when the hæmorrhage has been recent this does not hold,—though indeed, when it is of some days standing, a specimen is obtained without red cells but tinted with hæmoglobin derivatives to be recognized with the aid of the spectroscope. To sum up, one must grant that instances now and then occur in which it is impossible to state whether the blood present in the cerebrospinal fluid depends on a previous intracranial or intraspinal hæmorrhage, or on injury done in lumbar puncture. When operation is in question, much rests on clinical features and on the skill with which lumbar puncture was accomplished.

In the normal cerebrospinal fluid there are from 1 to 7 white cells per c.mm. Probably they are never entirely absent. When the meninges are sound lymphocytes alone occur, often much degenerated. On the whole the cell number is a remarkably sensitive index to the state of the meninges. As might be supposed, all gradations are seen, from a single lymphocyte in every 2 to 3 c.mm. to many thousand polymorphonuclear neutrophiles in a pus so thick that it can be evacuated only through a canula of large bore (pus much too thick to use in the "melangeur," which is, of course, unnecessary in the face of the gross appearance of the fluid). Speaking broadly, the purulent fluids, with 4,000 to 40,000 white cells per c.mm. and upwards, pertain to the acute meningeal infections; next, in point of cell-number, comes tuberculous meningitis, in which as many as 2,000 white cells per c.mm. may occur, with 200 to 300 c.mm. as an average count. A fluid containing several hundred cells per c.mm. may yet be clear macroscopically. Syphilitic meningitis, tabes, paresis, give usually more than 10 and less than 100 cells per c.mm. Trustworthy statistics on the subject are few. According to Fuchs and Rosenthal, who summarized the numerous investigations made with the technic of Widal, Sicard

and Ravaut, over 90 per cent. of the cases of syphilitic and parasyphilitic nervous affections exhibit during their acute stages an increase in the cell-content of the cerebrospinal fluid, whereas of those diseases which do not depend on syphilis but 6 per cent. of cases showed cell-increase. Thus it becomes possible to distinguish between paresis at its inception and other conditions with which it may be confused; and to pronounce on the question of tabes versus a neurosis. A "spinal lymphocytosis" has lately been observed to accompany the headache of secondary syphilis, and to occur sometimes in the course of epidemic parotitis and of herpes zoster, but precise statements on these points are lacking. On the whole the number of diseases in which the cell-content of the cerebrospinal fluid is increased are relatively few, and these fortunately are not of a sort to be confused. On the other hand, the maladies from which it is important to distinguish them only seldom yield a "spinal leukocytosis," so that the test is a cogent one. Further, there occur cases of acute infectious disease in which the organism may be cultivated from the cerebrospinal fluid in the absence of a meningeal process. Here the lack of a cell-increase, as determined by the count, alone enables us to affirm the absence of a meningitis.

It has been noted that when in a case with healthy meninges one lumbar puncture follows close after another the second specimen of fluid may show an increase in cells, perhaps to 100 per c.mm., some of them polymorphonuclears, whereas cytologically the first specimen was normal. Such a change is due to an injury produced by the needle at the first operation. The fact then that lumbar puncture may itself give rise to a slight, transient increase in the number of cells in the cerebrospinal fluid should always be remembered when one has to deal with a specimen from a patient, who, some few days previously, submitted to another lumbar puncture.

In general, should the subarachnoid pressure be normal and the number of cells in the fluid less than 10 per c.mm., it is unnecessary to proceed to a differential cell-count. Lymphocytes alone will be found, and these so few that they can be obtained in smear only after strong centrifugalization under careful technic. Even when 50 to 100 elements are in each c.mm. it may be difficult to obtain a satisfactory cover-slip preparation of them. This should be pro-

ceeded to promptly, since in the cerebrospinal fluid at room temperature the cells degenerate rapidly and in the course of six hours will often be unrecognizable, if they have not disappeared. To sediment them satisfactorily requires rapid centrifugalization for some 10 to 30 minutes. With the sediment of cells obtained, it is incumbent to follow a special technic in transferring it to the cover-slips; for it may be completely scattered if one attempts to draw it up into a pipette as one does a urinary sediment. Instead, the fluid is drained away from the cells by slowly and completely inverting the container. While it is still held upside down the slight portion of fluid left clinging in the bottom is taken up in a capillary tube, and, with the cells which it contains, is blown out upon cover-slips, dried, and stains duly made. One of the Romanowsky modifications (Hasting's stain, Wright's stain) is convenient for this purpose.

Lymphocytes (and perhaps an occasional large endothelial cell) are alone normal to the fluid. In a differential count from the stained specimen the headings Hastings suggests,—polymorphonuclear cells, mononuclear cells and endothelial cells,—may be adopted. For all purposes of diagnosis these are sufficient. Mast-cells have not yet been reported, and eosinophiles seldom in any quantity, though a warning must be given against the " pseudeosinophile " appearance taken not infrequently by the neutrophiles in any exudate. Tumor cells have been recognized, and, indeed, in rapidly degenerative processes, nerve-cells; but these are to be looked on as most exceptional, if not questionable. Mononuclear cells are common, from those of 30 to 35 microns to the small lymphocyte; and one French observer, measuring the latter, finds that with chronicity of the meningeal process their average diameter is less. Endothelial cells 50–60 microns across, with undifferentiated protoplasm, and a relatively small, vesicular, often eccentrically placed nucleus, occur, but are not confined to one affection.

Precise cell formulæ have been found of little value. That part of every cell formula; and, so far as we know, that part alone, which expresses the relationship between polymorphonuclear cells and those mononuclear, is important. The polymorphonuclear cells, speak, as in thoracic and peritoneal exudates, for an acute process; the mononuclear (the lymphocytes) for chronicity. Though the latter are prone to occur in cases of tuberculous meningitis they

are by no means limited to that malady. Smears from a case of epidemic cerebrospinal meningitis of three to four weeks standing often coincide cytologically with those from a newly appeared tuberculous meningitis. In this affection if it take an acute form, polymorphonuclear cells may predominate; though it is notable that here the cell-content in general is less than in meningitis of another bacterial origin, and only very rarely sufficient to render the fluid purulent,—a fact of diagnostic worth. In cerebrospinal syphilis and the parasyphilitic nervous diseases the mononuclear is always the prevailing cell, and (at least so far as the author's experience goes) the only one.

The fluid remaining from the cell-count and that supernatant after centrifugalization may be devoted to the protein determination. A globulin and an albumin have been recognized in the cerebrospinal fluid, the former always present, the latter, when at all, only in traces. The idea that a pathological increase in protein affects only the albumin has given way of late, as it has been recognized that the two are often increased together, and that occasionally the amount of globulin alone is changed. But the relation of these proteins has been so little studied that, for clinical use, the quantity of the two combined alone has significance. For this, the heat test as applied to the urine has some worth, but it demands that the observer acquaint himself with the amount of clouding obtained from normal specimens. To quantitate with acetic acid and potassium ferrocyanide requires 10 cc. of fluid and special apparatus. Simpler, though not so precise, is Esbach's test in one of its many forms, perhaps accomplished best, for the 4–5 cc. of fluid at hand, by the employment of a narrow test-tube, pencilled off into lengths to correspond with the Esbach tube for urine, as ordinarily sold. Width of bore in this instance does not influence the depth of the precipitate, providing that the quantitative relations between the picric acid reagent and the fluid to be tested remain the same. While it is true that the scale in grams as marked for urine albumin can have no application to the protein quantity, yet it will serve to relate the protein amounts in various specimens. The method is only tolerable because none as simple and more certain has been devised, and because, after all, it serves to show well the protein modifications. With it normal fluids in general show less than ½ gm. protein

(by scale) per litre; tuberculous meningitis always a larger amount, ¾ to 2 gm. ordinarily; and a "purulent meningitis" in its acute stage often so much protein as to clog the tube and prevent a reading. Syphilitic meningitis, paresis and tabes, according to severity and stage, may or may not yield a fluid with a pathological amount of protein. Protein-increase and cell-increase as a rule go hand in hand associated in the same diseases, but the relationship is not constant.

With protein, cell-content and cell-character of a specimen of cerebrospinal fluid and the subarachnoid pressure all known, one may be in a position to state much. First, as to the presence or absence of a meningeal affection: If the fluid be clear and colorless macroscopically, the cells less than 8 per c.mm. and the protein less than ½ gm. per litre by the Esbach test, the subarachnoid pressure between 50 and 300 mm. of the fluid itself, this may be considered as strong, and so far as is known, conclusive evidence against an active pathological change in the meninges. A red, yellow or brown fluid under increased pressure, showing erythrocytes, or, spectroscopically, hæmoglobin derivatives, accompanied by protein increase, speaks for hæmorrhage into the subarachnoid space. When tabes, paresis or cerebrospinal syphilis is suspected, a specimen showing moderate cell increase (10 to 200 cells per c.mm.), mononuclear in character, with or without protein increase, the pressure being normal, speaks for a chronic meningeal process, and should help much to confirm the diagnosis. When meningitis is in question, a definite increase in cells, especially if pressure and the protein amount are greater than normal, attests for it, even when the fluid seems limpid and is bacteriologically negative. Such a condition (the fluid clear with 50 to 500 cells per c.mm., ½ to 2 gm. protein per litre, pressure 200 to 600 mm. of the fluid itself) is often present in tuberculous meningitis, and enables one at once to state that a meningitis is present, and to decide as to its probable etiology. One must keep in mind, however, that the fluid from any chronic meningitis may present this picture. Cases of brain tumor and of hydrocephalus usually show a fluid itself normal to the tests given, but under much increased pressure. In uræmia a similar condition may obtain, except that the fluid contains urea. As regards specimens cloudy with cells or purulent,

while it is true that pressure and protein are increased, the macroscopic appearance and the bacteriological findings suffice for a diagnosis.

The fact that the majority of investigators have busied themselves so far with a single character of the cerebrospinal fluid, tracing out the influence on it of this or that disease, and that the results thus obtained have not yet been correlated, accounts for the comparative inexactness of our knowledge with regard to the interpretation to be put on the individual specimen. To gain the facts that will make this interpretation sure and will widen its application, standard or routine methods of examination of the cerebrospinal fluid, sufficiently simple for clinical use must be adopted.

Surgery

PERFORATED DUODENAL ULCER

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It is not very many years ago that duodenal ulcer was considered as very difficult of diagnosis, and was treated almost exclusively by medical measures. Surgeons only saw cases for one complication, viz., perforation, and even then they were often called unfortunately at a very late hour so that the results of surgical treatment were by no means good. Of recent years the subject has undergone a complete change: the interest in duodenal ulcer is now in great part surgical. The surgical aspects of duodenal ulcer have been prominently brought forward in this country by Robson and Moynihan, and in America by Murphy and Mayo. These surgeons, as well as many others, have done so much to increase our knowledge of duodenal ulcer from the surgical point of view that at the present day numerous operations are undertaken for the relief of this condition or one of its complications, and not perhaps the least frequent complication which the surgeon is called upon to treat is *perforation*. A reference to the cases treated by these surgeons shows how infrequently perforation occurs, compared to other complications for which they have operated. Perforation of a duodenal ulcer was never at any time, judging from literature, a very common condition, but it is probably not more rare at the present day than formerly, in spite of the many operations performed for the relief of a duodenal ulcer.

In the last three years I have operated upon 5 cases of perforated duodenal ulcer, and have seen 5 other cases operated upon besides 1 in which no operation was performed, thus making a series of 11 cases.

The notes of my cases are as follows:

Case I.—Male, aged forty-four, operated upon in March, 1904. He had always enjoyed very good health: it was impossible to obtain any history of previous gastric or intestinal trouble. Whilst lifting a heavy packing-case he was suddenly seized with acute pain in the right hypochondriac region; he became very faint and had to be carried to the hospital, a distance of about 100 yards. On admission to the hospital he had recovered to a great extent from his initial collapse; the pulse was of good quality, 80 per minute; temperature 97 degrees. He vomited three times in rapid succession; the vomit was bile-stained. He complained of considerable pain in the right hypochondriac region, and this region of the abdominal wall was held very rigid; the maximum point of tenderness was about 1 inch above and to the right of the umbilicus. Apart from the right hypochondriac region the abdomen was free from all tenderness and moved freely on respiration. No food had been taken for some two hours previous to the occurrence of the acute pain. Two or three hours later the pain was much easier; he again vomited some bile-stained material. The temperature had risen to 100.6 degrees and the pulse to 104 per minute. During the night he slept well and appeared free from pain.

The following morning he did not complain of any pain, but on deep pressure some tenderness was elicited in the right hypochondriac region. The temperature was 100 degrees, and the pulse 98 to 106. The abdomen was perfectly soft and moved uniformly on respiration; on percussion the abdomen appeared quite normal. Later in the day his condition changed, and rather suddenly. The pulse increased in frequency and reached 130 per minute; the temperature rose to 102 degrees; respirations were unaltered. The abdomen was slightly distended generally; abdominal movements were free, except in the right hypochondriac region, and there the wall was held rigidly fixed and great tenderness was complained of, the remainder of the abdomen being free from all pain and tenderness. On percussion there was an area of tympany reaching from the umbilical level on the right side upwards to the fifth rib in the nipple line, backwards to the mid-axillary and inwards to the midlines. There was absence of liver dulness anteriorly. When the patient was turned on to his left side this tympanitic area extended

backwards to the scapular line, beyond which level some impaired resonance was obtained over the right base where the entry of air was deficient. Some impaired resonance was also present in the right loin. These signs pointed to a localized collection of fluid and gas in the right hypochondrium. The diagnosis made was a perforated duodenal ulcer.

Operation.—Twenty-eight hours after the initial symptoms a median epigastric incision was made and lengthened eventually to about 5 inches. On opening the peritoneal cavity some turbid fluid escaped; this was coming from the right side; the remainder of the peritoneum did not appear to be infected and was, therefore, shut off by gauze packing. The duodenum just beyond the pylorus was considerably thickened and adherent to the liver; from amongst these adhesions fluid was issuing forth. The adhesions to the liver were separated and a perforated ulcer exposed in the centre of a very indurated duodenal wall; the perforation was about the size of a threepenny-piece. An attempt was made to close the hole with Lembert's sutures. The bowel, however, was too friable to permit this procedure. The free edge of the omentum was brought up and stitched around the perforation. The infected peritoneum was carefully dried. A counter-incision was made in the loin just below the last rib, and as soon as the peritoneum was incised here, some few ounces of turbid fluid escaped. Rubber drainage tubes were inserted through each incision, the anterior one being closed in great part. The operation lasted about twenty-five minutes. There was some collapse during the operation, and at the close a pint of saline solution was infused into the veins. The following morning the patient had a rigor and the temperature rose to 105 and the respirations to 36 per minute. Lung trouble was anticipated, but the alarming symptoms subsided in a few hours, and the temperature fell to normal in a few days. The tubes were removed on the eighth day. A few days later a small abscess developed in the abdominal wall, presumably due to stitch infection. This was treated and all went well.

For seven months after the operation I frequently saw the man. During this time he was in perfect health and suffered from no symptoms whatever. I then lost sight of him for a few months. At my request he came to see me about fourteen months after the

operation. He had suffered for some few weeks from "indigestion," —*i.e.*, pain occurring after food a variable time, and lasting about half an hour. His appetite was good; he had not vomited and never had any hæmorrhage as far as he knew. The stomach was not dilated. He was not losing flesh. This "indigestion" was probably due to the unhealed duodenal ulcer. Since that date I have lost sight of him.

Case II.—Operated upon in April, 1904. Male, aged thirty-seven.

He was thought to have had an attack of acute appendicitis two years previously, and the history he gave of this illness strongly suggested that the appendix was at fault. Apart from this, there was no history of gastro-intestinal disease. He was suddenly seized with acute abdominal pain, chiefly on the right side, and the maximum pain was in the right lumbar region. The pain occurred two hours after dinner; there was some collapse. Six hours later he was easier, but still had considerable pain in the right side of the abdomen. The abdomen moved fairly freely, with some restriction on the right side; the wall was somewhat rigid here, but not particularly tense in the right iliac fossa. Temperature 100.2 degrees; pulse 80–90. A few hours later the pain increased in severity, the pulse became more frequent, and the temperature was rising. When seen thirty hours after the onset he appeared very ill. The temperature was 102.6 degrees; pulse 120. The abdomen was somewhat distended and its movements were restricted. The whole of the right half of the abdomen was rigid; the maximum tenderness was in the right iliac fossa. There had not been any vomiting; the bowels had moved once. Liver dulness was present to the normal extent. The man was getting worse. From the previous attack of supposed appendicitis, the absence of all other previous symptoms, and, when seen, the maximum tenderness in the right iliac fossa, I made the diagnosis of perforative appendicitis. Pelvic examination was negative.

Operation.—Thirty-one hours after the initial symptoms an incision was made in the right iliac fossa; on opening the peritoneal cavity some turbid, odorless fluid escaped; no gas was noticed. The appendix was immediately exposed and found to be healthy. A gastric or duodenal perforation seemed therefore most probable,

and the abdomen was opened in the epigastrium. A considerable quantity of turbid fluid welled up from the right side. The stomach was normal, but on tracing the duodenum a small perforation was discovered about three-quarters of an inch beyond the pylorus. This was closed with a layer of Lembert's sutures. As the margins of the perforation were very unhealthy and did not hold the stitches satisfactorily the omentum was drawn up and stitched over the site of perforation. The peritoneum was then cleansed. The peritonitis appeared to be localized to the right side of the abdomen. A counter-opening was made in the right loin and a tube passed through this. The epigastric and iliac incisions were drained with gauze. The operation lasted forty minutes. The patient made an uninterrupted recovery. The gauze drains were entirely dispensed with on the seventh day, and the tube removed on the tenth day.

Four months later the man was in fair health. He was then suffering from what he called "indigestion,"—*i.e.*, occasional pain some two or three hours after food, which lasted a variable time. This was unaffected by diet. Presumably the pain was due to the unhealed ulcer. Since that time I have not heard from him.

Case III.—Operated upon in May, 1904. Male, aged fifty-three.

He had suffered from indigestion for three years. The symptoms of this indigestion was pain about half an hour after meals; it was not constant; he was often free for weeks together. The pain lasted about two hours and was often relieved by taking food. There was no vomiting; he suffered often from flatulence. For three weeks the pain had been getting worse and was particularly severe at night time; it then had no relation to food, and he would often take food to relieve himself. He was suddenly, whilst running after a 'bus, seized with acute abdominal pain and collapse; he was able to walk a few yards to a doctor's house, where he lay very collapsed for about an hour, being quite unable to move. He was then carried to a hospital, a few yards away. On admission he was evidently in considerable pain. Temperature 96 degrees; pulse 90 per minute; respirations 26 per minute. The abdomen was flattened, and moved only slightly on respiration; it was generally rigid and markedly tender in the right hypochondriac region. When I saw

him, about eight hours later, he was still in considerable pain; the abdomen was moving more freely, but there was still some tenderness in the right hypochondriac region. Temperature had risen to 99 degrees; pulse 90. No abnormal percussion changes were noticed. There had not been any vomiting. He was the subject of chronic bronchitis and emphysema, and had been under medical treatment for some months for bronchial asthma. From the history and clinical signs I suspected a perforated duodenal ulcer, and advised operation. This was absolutely refused. A few hours later he consented to an operation, and I again saw him. At this time his abdominal symptoms seemed to have disappeared; his pulse was 90 per minute and the temperature 99 degrees. I doubted my original diagnosis, especially when he informed me that he had frequently had attacks like this in the past few weeks. No food was allowed by the stomach. The pain in the right hypochondriac region, however, returned, and with it the abdominal rigidity. The pulse rose to 100 per minute and the temperature to 99.8 degrees. These facts, together with the severe initial collapse, justified the diagnosis of perforated duodenal ulcer and immediate operation.

Operation.—Twenty-two hours after the initial symptoms a median epigastric incision was made about 5 inches long. On opening the peritoneal cavity 3 or 4 ounces of turbid fluid escaped from the right hypochondriac region; the stomach and intestines in this region were coated with lymph. In the first part of the duodenum was a tiny perforation surrounded by very indurated bowel wall. Some sutures were passed to close the perforation, but nearly all the sutures cut out as soon as they were tied. The omentum was drawn up and sewn over the perforation with a few sutures. The peritonitis was localized; the peritoneum was carefully cleansed; a lumbar incision was made and a drain-tube inserted; a gauze drain was passed down to the site of perforation.

The following day he developed acute bronchitis which gave rise to considerable anxiety for a few days. This, however, gradually subsided. The wound sutures gave way and caused considerable gaping of the edges. The lumbar wound healed in a fortnight; the epigastric sinus discharged for some two or three weeks longer, and the wound was very slow in healing. It eventually healed with a firm mass of fibroid tissue.

After the operation, for some few weeks, all his gastric symptoms left him; he had no pain and he could eat every kind of food. Later he began again to suffer from pain after meals, of variable time and of variable duration; flatulence returned, and, as far as his abdominal symptoms, he relapsed into just the same state as before operation. Occasionally he is free from all symptoms, but for some days together he suffers, at times, from severe indigestion.

Case IV.—Operated on in November, 1906. Male, aged forty-five.

Has generally been a healthy man. About eighteen months ago he had a short period of "indigestion." For a few days he suffered severely from pain coming on about an hour or so after food and lasting an hour or two. Diet made no difference in the severity of the pain. He did not vomit, and he never noticed any melæna. Then followed an attack of severe abdominal pain of sudden onset. He was somewhat collapsed, and was confined to bed for two days and was carefully dieted. The diagnosis made was colic, but in all probability it was a local peritonitis around a duodenal ulcer. From this he recovered completely and had not suffered any pain until three days previously. Three days ago some pain was again experienced, coming on about an hour after food and lasting about an hour. The pain he describes as of a "cutting" character. It was situated in the mid-epigastric region and radiated around the right hyponchondriac region to the back. He has been at work during the three days. The pain, he says, is as severe after taking fluids as after solids.

Whilst driving home in his cart on November 3, 1906, he was suddenly seized with severe abdominal pain, and fell forward in the cart. He just managed to stop the horse and called for help from some passer-by who drove him home. When he got home he had a little stimulant, as he was so cold and faint; otherwise he had not taken any food for three hours. Soon afterwards he was taken to the hospital. On his arrival there, about four and a half hours after the severe pain, he was still extremely collapsed, and looked like a dying man, and was complaining of very severe pain in the abdomen. On the administration of restoratives he recovered somewhat from his shock. He had not vomited up to this time. His abdomen was very hard and rigid. Temperature 97.2 degrees;

pulse 62. A diagnosis of perforated duodenal ulcer was made. I saw him six and a half hours after the commencement of his pain. He then complained of severe pain in the upper region of the abdomen, especially in the right hypochondriac and right lumbar regions; the whole abdomen was motionless; on palpation it was rigid in the epigastric and right hypochondriac regions, but elsewhere was comparatively soft. On percussion there was an area of tympanitic resonance in the right hypochondriac region obliterating the liver dulness; this tympanitic note extended upwards to the fifth rib and downwards to the umbilical line; on turning the patient on to his left side this area of tympany was noticed to extend well round to the left side. (Two hours before this the abdomen had been carefully percussed and no change from the normal had been noticed.) Temperature had risen to 98 degrees; pulse 90. He had vomited some bile-stained mucus with streaks of blood twice since admission.

Operation.—Six and a half hours after initial symptoms a median epigastric incision was made, and on opening the peritoneal cavity some thin watery fluid issued up from the epigastric region; this was non-odorous. An examination of the stomach revealed nothing. On tracing the pylorus and duodenum a sudden gush of bile and gas was noticed; this was wiped away and a perforation in the duodenum, about ½ inch beyond the pylorus, on the anterior surface, was seen; it was about ¼ inch in diameter, and its margins were only slightly thickened. The perforation was closed with interrupted sutures and then with a continuous layer over the first row of stitches. The walls of the intestines did not hold the stitches satisfactorily and so some omentum was turned up and sutured over the site of perforation. There was very little escape of material into the peritoneal cavity; the latter was wiped dry; a lumbar incision was made and a drain-tube inserted from here; the epigastric incision was drained with gauze. During the operation there was some collapse and the patient had to be stimulated. For some time after the operation he was collapsed and required constant care; strychnine was given frequently. He was fed in forty hours, as there had not been any sickness. Bowels opened on the second day. The epigastric wound discharged very slightly, but the lumbar one discharged profusely for a few days. The gauze was removed on the

fifth day and the tube dispensed with on the eighth day. The wounds were completely healed on the sixteenth day. The temperature ranged from 98 to 100 degrees the first few days and after that fell to normal; the pulse never was more than 90 per minute after the operation.

After the operation the patient was carefully dieted for the ulceration. In spite of this, one month later, he was suffering from epigastric pain after his food, and from some tenderness. These symptoms are no doubt due to the ulcer. I have quite recently seen the patient. His pain still persists.

Case V.—Operated upon in 1905.

A case of suphrenic abscess due to the perforation of a duodenal ulcer. Male, aged twenty-six. He had always been a healthy man. No note was made of any gastro-intestinal disorder. This was not asked for directly, since the real origin of the disease was not discovered until the autopsy.

A week before admission to the hospital (April 13) he was apparently quite well save for some constipation, and for this he took some magnesium sulphate. This caused the bowels to move three times in rapid succession and he felt very "uncomfortable" in the abdomen for the remainder of the day. As he was walking home from work in the evening he was suddenly seized with acute abdominal pain over the whole abdomen; he had not had any food for some hours previously. He went to bed immediately on getting home and had severe pain the whole night. He was not sick. The following day the pain persisted and was especially experienced in the right hypochondriac region. For the following few days the pain was still present but less in severity. He was kept on fluid diet, but he had no appetite. He remained in bed. He was said to have had some fever the whole time, but no accurate note of his temperature and pulse was kept. During the illness the bowels acted very freely and were of a blackish color (probably due to the presence of blood).

Admitted April 20. He was then in some pain which he located in the right hypochondrium and in the right lower chest. Temperature 102 degrees; pulse 120 per minute; respirations 28 per minute.

The *abdomen* was not distended; it was generally soft and

supple. The liver was palpable well below the costal margin, and on deep pressure over it considerable tenderness was elicited; the liver descended only slightly on respiration; the respiratory movement of the upper abdomen was somewhat deficient.

The *chest*: The heart's apex-beat was displaced upwards and outwards, and was felt just under the left nipple. There was no œdema around the chest. Dulness was present anteriorly up to the fourth rib and around the side of the chest, and posteriorly to a level corresponding to this point. This area of the chest was quite dull, and some impaired resonance was present above this posteriorly to half-way up the scapula. The breath sounds were absent over the dull area, as was also the fremitus and vocal resonance. The area of dulness and the limit of the breath sounds were not altered by any change of position of the patient. On turning the man on to his left side some friction was heard just above the lower angle of the scapula.

During the three following days the physical signs remained about the same; the temperature ranged from 99.2 to 102.8 degrees; the pulse from 100 to 120; the respirations increased on the day after admission to 40 and two days later to 52.

April 24. Four days after admission a needle was inserted into the right chest and pus withdrawn; he was then transferred to the surgical wards with the diagnosis of empyæma. Under anæsthesia a needle was inserted in the ninth space in the posterior axillary line, pus was withdrawn, and a portion of the ninth rib was resected; about 2 pints of foul pus was evacuated. On further exploring the cavity from which the pus came, it was eventually discovered that it was below the diaphragm, between this and the liver. A drain tube was inserted into this cavity. The temperature fell somewhat after the operation, but his general condition did not improve. The temperature rose again and ranged from 98 to 102 degrees; the pulse increased in frequency and the respirations varied from 40 to 56 per minute. Death occurred on the third day.

Post-mortem.—The abscess cavity was situated between the diaphragm and liver, and bounded below by the hepatic flexure of the colon, and the adhesion of this and the anterior border of the liver to the abdominal parietes. The duodenum was firmly bound to the inferior surface of the liver, and on separating these dense

cicatricial adhesions, a perforation of the duodenum was seen in front. The ulcer was a chronic one, was situated 1 inch beyond the pylorus on the anterior superior angle and was ¾ inch in diameter. The inferior, right, and part of the superior surface of the liver formed part of the wall of the abscess cavity. The diaphragm was pushed up to the fourth rib in the mid-axilla.

Pleuræ.—Left side nil. Right side contained a pint of purulent fluid.

Pericardium contained 1 ounce of turbid fluid.

Lungs.—Left, severe œdema. Right, partly collapsed; no pneumelæna.

The following cases, not under my personal care, I have had the opportunity of seeing.

Case VI.—Male, aged forty-four. For three years he suffered with severe indigestion; the pain usually, but not always, occurred after food, and was often very severe; no vomiting; he never noticed melæna.

On February 9, 1899, he was suddenly seized with severe and acute pain in the epigastrium after a supper consisting of sandwiches and cocoa; vomiting set in and persisted. Admitted the following day. He was then very collapsed; pulse 120 per minute; temperature 98 degrees. The abdomen did not move with respiration; it was very rigid but not distended; the maximum pain was around the umbilicus.

Diagnosis.—Perforation of gastric or duodenal ulcer.

Operation.—Nineteen hours after onset of symptoms. The peritoneal cavity contained a quantity of bile-stained fluid, with very little lymph; the whole peritoneum seemed involved. There was a small perforation, ¼ inch in diameter, just beyond the pylorus; this was closed by two layers of sutures. The peritoneal cavity was irrigated. He was very collapsed during the operation. He never rallied from the shock and died fourteen hours later.

Post-mortem.—Acute general peritonitis; the effusion was chiefly between the stomach and the liver, and the liver and diaphragm. The sutures securely closed the perforation.

Case VII.—Male, aged twenty-two. For some time he had had occasional attacks of indigestion. On May 5, 1900, he was suddenly seized with violent abdominal pain. He went to a dispensary

near, and was given some medicine; whilst there he was so collapsed that he had to be carried home. The following morning the pain was still severe and vomiting commenced. His bowels had not been opened. The following day he was worse, and was sent to a hospital.

On admission he was evidently very ill; he was flushed and sweating; his breathing was rapid and shallow; pulse 120 per minute. The abdomen did not move on respiration, it was distended and generally gave a tympanitic note; the maximum tenderness seemed in the right iliac fossa.

Diagnosis.—Perforative appendicitis.

Operation about forty-eight hours after the onset of the severe symptoms. Incision was made along outer border of right rectus in iliac region; quantity of undigested milk was free in peritoneal cavity; incision prolonged upwards and a perforation found in duodenum; this was closed by sutures. The peritonitis was extensive; the peritoneal cavity was irrigated and drained. He rallied from the shock of the operation, but the following day vomiting set in and persisted; he became very collapsed and died.

Case VIII.—Male, aged twenty-eight. For three weeks previously had had severe pains in the epigastrium after food. He was suddenly taken with very severe abdominal pain and collapse, and was *brought* to the hospital.

On admission he was too ill to obtain any satisfactory history. His eyes were sunken; pulse 130 per minute.

The abdomen hardly moved at all; it was distended, generally tympanitic; the liver dulness had disappeared; there was universal tenderness. He was vomiting freely.

Diagnosis.—Perforative appendicitis.

Operation.—Exact time doubtful, but well within twenty-four hours, and probably not more than twelve hours, after perforation. On opening the abdomen below the umbilicus, gas escaped in some quantity with a loud hissing noise; this was not offensive; the abdomen then collapsed and some turbid fluid escaped. The appendix was normal. The abdomen was opened above and a perforation of duodenum found just beyond the pylorus; the perforation was round and clean, and the edges were considerably thickened. The ulcer was circumscribed and cut away, and the wound in the duo-

denum closed. The peritonitis was general and the pelvis was full of turbid fluid; the peritoneal cavity was irrigated. He never rallied from the shock of the operation and died shortly afterwards.

Post-mortem.—The peritoneum was generally intensely injected, but practically free from fluid. There was some narrowing of the duodenum at the point of suture.

Case IX.—Male, aged forty-five. About two years ago he had some pain, especially in his back, and he vomited about half a pint of dark blood. For a long time he has been subject to morning sickness and can only take a cup of tea for his breakfast. He had had diarrhœa attended with some pain in the abdomen for about two years, evacuating watery stools three or four times daily; he had noticed his stools at times as dark as coffee-grounds. His appetite had been impaired for the last two years. He drank on the average about 5 or 6 pints of beer a day.

On September 6, 1904, he was suddenly seized with acute abdominal pain which came on immediately after dinner; he went to a chemist's shop, which was quite near, for some medicine to relieve the pain. The pain became so severe that he was led by a friend to the hospital, a distance of about a quarter of a mile away. He had not vomited. On admission he complained of severe pain, griping in character and constant in position, about 2 inches above and to the right of the umbilicus. The pain was continuous, with frequent exacerbations which were severe enough to make him groan; the pain did not shoot in any direction; it was worse on deep breathing. Temperature 98 degrees; pulse 96. There was extreme tenderness and board-like rigidity of the upper half of the abdomen; the rest of the abdomen was also somewhat rigid; the percussion was normal. The following morning he looked and felt better; temperature 98 to 100 degrees; pulse 100. Later in the day the liver dulness had disappeared, but without any distention of the abdomen. On turning patient on to his left side the phenomenon of "shifting gas" made out over the liver area.

Diagnosis.—Perforated duodenal ulcer.

Operation.—About twenty-eight hours after initial symptoms. On incising the peritoneum free gas escaped, and some turbid fluid from the right half of the abdomen. A perforation was found in the duodenum, and around the perforation the bowel wall was

tough and indurated. The perforation was closed with sutures (two layers). The inflammation seemed limited to the right half of the abdomen, and this part was dried and two drains inserted, one at the site of perforation and one to the right renal pouch. On the third day after the operation some bile escaped from the upper tube; this ceased in a few days and the wounds healed in about five weeks.

Four months after the operation he looked very well; he could eat anything and everything; he occasionally had some pain in the right epigastric region, but this had no relation to food and food did not ease it. A recent letter from him tells me that this is his present condition. He occasionally has some pain, but he does not worry about it.

Case X.—Male, aged thirty-six. He had not had any previous indigestion of any kind; he felt perfectly well until to-day. About six weeks previously he had passed some tarry motions for three or four days, unaccompanied by any other signs. On March 2, 1905, he was suddenly seized with violent pains in the abdomen; he was not making any effort at the time; he was not sick. The pain continued, and was most marked in the right hypochondrium. Admitted one hour later. Temperature 97 degrees; pulse 96 per minute. Complained of severe pain in the right hypochondriac region; abdomen moved little on respiration; it was rigid and retracted; breathing short and rapid, evidently painful; liver dulness was present to its normal extent. Later in the day, temperature 100 degrees; the pain continued; the liver dulness anteriorly was replaced by a tympanitic area; this did not vary with movement of the patient.

Diagnosis.—Perforated duodenal ulcer.

Operation.—Seven hours after the initial symptoms. On incising the peritoneum some free gas escaped; very little fluid; perforation of duodenum ½ inch beyond the pylorus; this not reached satisfactorily through the median incision, and so a transverse one was made across the rectus. The edge of the perforation was hard and infiltrated; it was closed with two layers of sutures, some of which cut out and had to be replaced; some fat from the neighborhood was sewn over the perforation. One drain to site of perforation and one to the right renal pouch were inserted. The patient

made an uninterrupted recovery save for some stitch suppuration. He was seen two months later and was then in good health. He could eat anything, and he had not suffered from any pain since the operation. Since that date he has not been seen.

Case XI.—*Perforation; hæmorrhage; sudden death; no operation.*

Male, aged seventy-seven. Whilst walking along the road he was suddenly seized with acute abdominal pain; he was at once brought to the hospital. On admission he complained of pain all over the abdomen; the abdomen was generally tender without any localization; it moved fairly freely on respiration. He was only slightly collapsed; temperature and pulse were normal. A simple enæma was given, and after this the man was much relieved; his pain practically disappeared and he slept well during the night. Early the next morning he became suddenly collapsed, shortly afterwards passed a large quantity of blood per rectum and died within a few minutes.

Post-mortem.—The peritoneal cavity contained about a pint of dirty brownish-green fluid; this had escaped from the duodenum. The duodenum was partially adherent to the under surface of the liver, and about 1 inch from the pylorus was an ulcer which had perforated; the perforation was about ¼ inch in diameter, circular in outline with a sharply defined edge; the edges of the perforation were indurated. The stomach was normal; the liver and gall-bladder were normal; the pancreas showed a number of recent hæmorrhages; the kidneys showed chronic granular change.

The previous history was not inquired into in this case.

PREVIOUS HISTORY

In 4 cases of the series no previous symptoms of duodenal ulcer are recorded. It is only fair to say that in Case V and Case XI this was not directly asked for since the real disease was not suspected. Excluding these two cases, previous symptoms were present in 7 out of 9 cases. In one of the exceptions there was a previous history of an illness suggesting appendicitis rather than duodenal ulcer, but it is conceivable that the illness may have been due to the latter condition. In 2 cases hæmorrhage from the bowel was the only definite symptoms. This suggests a duodenal rather

than a gastric ulcer. In Case III the pain occurred about an hour after eating, lasted about two hours and was relieved by taking food. This was perhaps the most characteristic history of duodenal ulcer in the series. In the other cases I estimate that it was practically impossible to say whether the ulcer was gastric or duodenal from previous symptoms.

EXCITING CAUSE OF PERFORATION

By this I mean any strain or other condition which might have caused the perforation.

Case I, was lifting a heavy packing case; food two hours previously.

Case II, occurred two hours after dinner.

Case III, running after a 'bus.

Case IV, was driving a cart; no food for three hours.

Case V, was walking home; no food for some hours previously.

Case VI, immediately after sandwiches and cocoa.

Case IX, immediately after dinner.

Case X, was not making any effort at the time.

Case XI, walking along the street.

Only 1 case was under any severe strain at the time; 1 was running; 2 were merely walking; and 1 was driving. The relation of perforation to the ingestion of food is very inconstant.

DIAGNOSIS

In all cases there was some collapse at the onset. Generally the collapse was severe. In some cases it was extremely serious, and in one case it was so marked that the man was thought to be dying. Sometimes the patients were able to walk some little distance, but as a rule they were too ill to do this unaided. The duration of the collapse varied. There was always a period of reaction, except apparently in Case VIII. During this period there were practically always some symptoms present, but these were often not of sufficient severity to warrant the diagnosis of perforative peritonitis. Cases I, II, III, and IX showed very long latent periods. An examination of these patients during this period always showed some symptoms present. Sometimes it was fixed and constant pain and tenderness; sometimes pain was less marked, but some slight

rise in the temperature and pulse frequency were present. The pain was sometimes so slight in this stage that the patients had a comfortable sleep. The presence of gas in the peritoneal cavity was detected sometimes during this period without any further alteration in the general condition. These cases, at the operation, all proved to be examples of local peritonitis only, but also it was shown that limiting adhesions were absent, and hence the peritonitis would have become generalized. This series shows four cases in which the peritonitis was extensive, and in three of these there was a very short latent period; in the fourth case reaction from the collapse occurred promptly and serious symptoms remained in abeyance; although there was a pint of extravasation diffused over the peritoneal cavity, there was little evidence of such extensive peritonitis; death occurred in great part from hæmorrhage. In general the longer the latent period, the more local was the peritonitis. The cases serve to show that in any patient suddenly smitten with collapse and abdominal symptoms, even though reaction soon sets in and the symptoms seem *almost* entirely to have disappeared, a diagnosis and prognosis should never be hastily made until a sufficient interval of time has elapsed. The latent period in a perforated duodenal ulcer may perhaps be longer and be attended with fewer symptoms than any other perforative peritonitis.

Two cases of the series were mistaken for appendicitis on account of the maximum pain and tenderness in the right iliac fossa, and in these two cases there was nothing in the history to point to the duodenum. One was operated upon twenty-two hours and the other forty-eight hours after the first symptom. A third case was thought most likely to be of appendicular origin. In this case there was no symptom pointing to any definite viscus, and the appendix was considered as the most likely cause of the peritonitis. This case was operated upon about twelve hours after the initial symptoms. This confusion has often arisen. The distance between the duodenum and the appendix is quite small, and the extravasated contents may easily flow downwards to the duodenum, being directed there by the transverse mesocolon, the mesentery and the ascending colon. The exact site of perforation of the duodenum, previous adhesions, distention or collapse of the colon, and the movements of the patient are no doubt accessory

factors in determining the direction of flow of the extravasation. In the ultra-acute cases with overwhelming collapse and without previous symptoms, the diagnosis must remain unknown. There is nothing in these cases to point to the duodenum. Such a condition is unusual in duodenal perforation.

Five cases of the series were diagnosed correctly. The reasons for the diagnosis were that the symptoms—pain, tenderness and abdominal rigidity—were directed to the right hypochondriac and right lumbar regions. In 4 of these cases an escape of gas into the peritoneal cavity was demonstrated by clinical signs. This gave rise to characteristic resonance over the liver area, obliterating in whole or in part the normal liver dulness. The limits of the gas-containing cavity altered slightly on the position of the patient in 3 cases. In all these cases the abdomen had been previously percussed and the liver dulness found to be normal. The clinical signs of gas were developed more or less suddenly without any noticeable distention of the abdomen in this region, and at variable times in the course of the disease—2 cases at six to seven hours and 2 cases not till twenty-four hours after perforation. In 4 out of the 5 cases there were previous symptoms pointing to gastric or duodenal disease.

In 1 case a diagnosis of perforated gastric or duodenal ulcer was made upon the previous history. In this case the maximum pain, tenderness and rigidity were around the umbilicus, and not in the right side of the abdomen.

In the 5 cases which were correctly diagnosed the signs all pointed to a lesion in the right upper portion of the abdomen. This is more likely to result from a duodenal than from a gastric perforation, although, of course, a gastric perforation may give rise to similar signs. An examination of a larger series of cases shows that the signs of a duodenal perforation are not always manifested chiefly in the right side of the abdomen, but are not uncommonly more or less generalized over the abdomen, as in Case VI. The diagnosis then rests rather upon the previous history, and whether the previous symptoms are those of gastric or duodenal ulcer. The differential diagnosis of gastric and duodenal ulcer is often very difficult and at times impossible. The sex and age of the patient probably help but little in the diagnosis. The character of the pain

is the same in both. The time of the pain in relation to food, its relief or not on taking food, its exact situation, melæna without hæmatemesis, areas of tenderness and cutaneous hyperæsthesia are suggestive points. But in spite of all, the differential diagnosis must often be impossible.

In one case recently under my care I mistook a perforated gall-bladder for a duodenal perforation. The case was that of a man aged seventy-five years. He had been known to his medical attendant for some years and was the subject of chronic bronchitis. He had never had any symptoms of gastro-intestinal disease, or disease of the gall-bladder. He more or less suddenly developed pain in the abdomen, particularly centred around the umbilicus; there was little collapse; he vomited once or twice. The following day the pain was still present and very severe; he vomited incessantly; his abdomen became a little distended; temperature 99.6 degrees; pulse 100 per minute. On examination, some thirty-six hours after the onset of the pain, his abdomen was somewhat distended, moved very little on respiration and was generally tender, but particularly so in the right hypochondriac and right lumbar region. A diagnosis of peritonitis was readily made on general grounds. The cause, I thought, was probably a duodenal ulcer, as most of his symptoms were referred to this region. The case was one of empyæma of the gall-bladder. The gall-bladder was only slightly, if at all, enlarged; it contained pus and one gall-stone in the neck; the stone was freely movable; the perforation had occurred around the stone.

PROGNOSIS

The prognosis must depend upon many factors. The interval between perforation and operation must have something to do with the prognosis. But this is not the only thing. For in this series there are cases of recovery at 28, 31, 22, and 28 hours, and fatal cases at 12 and 19 hours after perforation. The extent of peritonitis is the most important factor. In three cases in which the peritonitis was more or less generalized a fatal result followed. In the successful cases the peritonitis was limited. Why in some cases, comparatively early, the peritonitis is diffused, and in other cases after some hours it is still more local, is not quite clear. I have drawn up a table of the cases for ready reference, and a study of

this shows that in these cases, in general, the interval between food and perforation, the food taken after perforation, the presence or absence of vomiting after perforation, and the movements to which the patient is subjected after operation bear no relation to the extent of the peritonitis. The latter must, therefore, depend upon other factors. The size of the perforation in the duodenum must vary only slightly and insignificantly, and cannot affect the prognosis. The presence or absence of previous adhesions may have some influence. It seems that the most important factor in the prognosis must be the virulence of the infection and the resisting power of the individual. This, judging from this series of cases, must be of more importance than the actual time of operation after perforation and other factors which are found in the accompanying table. The bacteriology of the peritonitis of a perforated duodenal ulcer is still more or less unknown. But upon this probably the prognosis depends more than upon anything else. The resisting power of the individual plays some part. His power to exude leucocytes and to form protective lymph are in all probability equally important factors. (See Table of Cases, page 109.)

TREATMENT

Ten cases were operated upon; 9 of these for non-limited peritonitis, and 1 for a localized subphrenic abscess.

The incision made in the cases diagnosed before operation was the median epigastric one. In 2 cases the abdomen was opened in the mid-subumbilical line first on an erroneous diagnosis; in 1 case the incision was made along the outer border of the right rectus on the diagnosis of appendicitis, and then was prolonged upon this line, and the duodenum was reached through this incision. The best incision from which to reach the duodenum is, no doubt, through the right rectus in a vertical direction. If one be confident of the diagnosis this incision should be made. But this does not give very good access to various parts of the stomach. I venture to think that the diagnosis of a perforated duodenal from a perforated gastric ulcer is, by no means, obvious, and I should recommend an incision just to one side of the mid-line, so as to divide the layers of the sheath of the rectus separately and suture them separately. In several cases of this series some difficulty was experienced in sutur-

TABLE OF CASES

Case	Interval between Food and Perforation.	Diet taken after Perforation.	Vomiting after Perforation.	Nature of Movements after Perforation.	Interval between Perforation and Operation.	Extent of Peritonitis.	Result.
1	Food taken two hours before	Fluid diet	Vomited three times	Carried to hospital almost immediately—about 100 yards	28 hours	Right renal pouch region and around liver	Recovery.
2	Two hours after dinner	Fluid diet	No vomiting	Unknown	31 hours	All the right side of abdomen, not the pelvis	Recovery.
3	Unknown	Nothing taken	No vomiting	Taken to a doctor's house quite near and then carried to hospital a few yards away	22 hours	Right renal pouch and pyloric regions	Recovery.
4	Three hours before	Only a little stimulant	Vomited once	Driven home and then to hospital, about a mile	6½ hours	Limited around duodenum	Recovery.
5	Some hours before	Fluid diet	No vomiting	Went straight to bed and stayed there for 7 days	11th day	Right subphrenic abscess	Death.
6	Immediately after sandwiches and cocoa	Unknown	Persistent vomiting	Admitted the following day	19 hours	Generalized	Death.
7	Unknown	Unknown	Frequent vomiting	Admitted 2 days after onset	48 hours	Extensive	Death.
8	Unknown	Nothing	Frequent vomiting	Brought almost at once to hospital	12 hours (?)	Generalized	Death.
9	Immediately after dinner	Fluid diet	Not noted	Went to chemist's shop a short distance, and then led by friend to hospital	28 hours	Right renal region and around liver	Recovery.
10	Not known	Nothing	No vomiting	Brought to hospital one hour later	7 hours	Right renal region around duodenum	Recovery.
11	Not known	Nothing	No vomiting	Brought to hospital almost at once	10 hours between perforation and death	A pint of fluid diffused over peritoneum	Death.

Table of cases showing that the interval between food and perforation, the food taken after perforation, the presence or absence of vomiting after perforation and the movements to which the patient is subjected after perforation bear no constant relation to the extent of the peritonitis.

ing the duodenum through this incision; and then it may be necessary, as in one case, to divide the rectus transversely.

It is of the utmost importance to close the hole in the bowel. A study of the literature of the cases in which the duodenal perforation was not closed shows a very high mortality. In one case of this series some bile escaped from the wound on the third day, but fortunately this soon ceased. The bowel around the perforation is often indurated and friable, and does not retain sutures well. In all my cases I have drawn up some omentum (and in some other cases in this series it has also been done) to close over the suture line; and in some of my cases I have relied almost entirely on this, with satisfactory results. The omentum should be sutured in position or else it will become disturbed by intestinal movements, or displaced on withdrawing the tube or gauze.

The treatment of the peritonitis in this series has not been uniform. In the cases in which peritonitis seemed to be generalized the whole cavity was irrigated freely. The number of cases is obviously too small from which to draw any conclusions. In the majority of cases the peritonitis was localized to the right upper half, or more extensively to the right half of the abdomen. Drying of the peritoneum with drainage seemed sufficient, as was borne out by the results. In these cases, quite apart from the teaching of the work of Dudgeon on the bacteriology of peritonitis, irrigation was absolutely contraindicated since the exudate appeared quite localized. Drainage was always employed, sometimes simply through the epigastric incision, but in all my cases I made a lumbar incision, and from this I found the greater part of the after-discharge came away. I think also that the lumbar incision, in some cases, caused a more speedy union of the epigastric incision and hence a more secure one. The exudate at first is quite thin and will easily flow through the tube, differing from some other forms of peritonitis. There has always been some discharge through the drainage. In no case has abscess formed which requires a further operation. In the first few days there has generally been a little fever, but this has subsided.

The after-history of the cases which recovered is as follows:

Case I.—For seven months he was perfectly well; later developed "indigestion" which was probably produced by the unhealed ulcer.

Case II.—Four months later he was in fair health; suffered from "indigestion" which was probably due to the unhealed ulcer.

Case III.—Irregular attacks of subacute indigestion as before the operation.

Case IV.—Operated upon recently. He has now some pain on the right side soon after meals.

Case IX.—Occasionally has some pain, but does not "worry" about it.

Case X.—No symptoms at all two months after the operation; since he has not been heard of. This patient had no pain before operation; his sign of ulcer was hæmorrhage per rectum.

Of these 6 patients who recovered 5 may be said to have suffered from symptoms of ulcer after the operation. Two of these 5 patients did not appear to have had symptoms previous to perforation. In all cases the ulcers were of the chronic type at the time of operation. In no case was gastro-enterostomy performed at the same time as the suture of the perforation. This is advocated and is done by some surgeons in some cases. In these cases it seemed quite sufficient for the patients to stand the shock of the one operation, without submitting them to a further and somewhat prolonged manipulation and risking a further infection of the peritoneum. They are all illustrations now, no doubt, of cases of chronic duodenal ulcer. In no case has duodenal stenosis been detected clinically.

SURGICAL SYPHILIS

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Lues is a disease which has become so extensive since it first made its appearance, that at the present time there is not a single section of the globe exempt from it. We know at present that syphilis is an infectious disease, the infection being derived from direct contact of one subject with another, or by indirect contact. In order that infection may occur, a small abrasion, or some breach of continuity in the integuments must exist, serving as a point of entrance for the virus and allowing it to enter the circulation.

Syphilis is a disease which in many instances is absolutely curable. The greatest difficulty of the tertiary period, and these are the cases which will be considered in this paper, are those instances which have entirely, or partially, lacked a proper course of treatment.

The most variable types of lesion may develop during the progress of syphilis; at first the integuments, mucous membranes, fasciæ, periostium, bones, cartilage, muscles, parenchymatous organs, nervous system, and organs of sense become involved. In point of fact, there is no organ of the body which may not at some time be attacked during the evolution of the disease.

Latent periods, extending over months or years, during which the patient feels perfectly well, and without giving any evidences of any activity on the part of the process, are met with; so that the patient believes he has been cured, although these periods of latency may alternate with those of relapse of indefinite duration, these relapses recurring throughout a number of years, or even during the whole life of the patient. From the fact that lues is encountered at all ages and not infrequently may give rise to symptoms which are far from being characteristic, it at once becomes

evident that the diagnosis of the real condition of affairs may be rendered extremely difficult. This is especially so, if a correct history cannot be obtained. It may be that the patients deny syphilitic infection, either from shame or from absolute ignorance, or again they may possibly forget. Then again, they not infrequently think that the trouble for which they consult the surgeon cannot be the consequence of the former infection, because since that time no evidences of the disease have been noticed, and, therefore, they do not think it necessary to refer to their former trouble. They are very apt to omit mentioning this, more especially when they are able to refer to some traumatism as a cause for their surgical affection.

In such instances, should all clinical evidences be wanting, such as enlargement of the lymph-nodes, cicatrices on the genitalia, leucoderma (which Kaposi and other authorities do not consider as characteristic diagnostic evidence, or specific products of syphilis, but only as accidental manifestations of local infiltration produced by syphilis), then it is not at all surprising if an erroneous diagnosis is made. It is quite certain that tumefaction of the inguinal, cubital, submaxillary, and occipital glands have very slight diagnostic value, because indolent, regional, or general glandular enlargement may occur from various causes and dyscrasias. Then again, they are of trifling diagnostic value, because they are very prone to disappear with the subsidence of other manifestations, although the patient may have presented a true syphilitic infection. However, enlarged glands may aid in the diagnosis when the symptoms present are doubtful, but their absence is far from being a proof to the contrary.

Among those cases most difficult to diagnose are certainly instances of late syphilis, especially those neoplastic lesions to which it gives rise, or its manifestations in the bones and joints. After these various affections have resisted every known method of treatment, the patient is placed upon antisyphilitic remedies as a last resort, and these lead to a correct recognition of the affection. In those cases also, where the patient has been exposed to infection at some former time, or where the attending physician has suspicions that syphilis may be in play, the diagnosis cannot be made with certainty, because antisyphilitic treatment may fail, when in reality

the lesion is due to lues. Then, again, it occasionally happens that a positive result from the treatment is not awaited, for the fear that the favorable time for operating may be missed. In these cases the nature of the disease is not infrequently recognized microscopically, after the patient has gone through a serious, or even a very mutilating surgical interference, thus showing that the operation was unnecessary. But the reverse is often true, instances of which I have related in several former publications.

The early teachings of Esmarch should not be forgotten, for long ago he published very instructive cases, showing the great danger of an erroneous diagnosis, calling particular attention to mistaking syphilitic neoplasms for malignant tumors, in order to prevent unnecessary and occasionally even dangerous operations.

As an illustration of an erroneous diagnosis I may mention two instances recently published by Rosenberger.

Although there is no viscus in the body which is exempt from syphilis, there are some organs which are especially liable to be attacked by the process. If, as a result of syphilis, the visceral organs become involved, the liver is in all probability included in the general process. Nevertheless, this organ may be the only one the seat of a lesion, in which case two principal forms are to be distinguished; namely, (1) diffuse interstitial syphilitic inflammation and (2) circumscribed hepatic syphilis, or hepatic syphiloma. These two forms are prone to occur simultaneously. The second form is of more especial interest to the surgeon, because, on account of the accompanying symptoms, the diagnosis of malignant new growth is easily suggested and leads to surgical interference, which, although not endangering the life of the patient to any great extent, is, to say the least, a useless bit of work.

As to the location of hepatic syphiloma, it may be said that they may occur at any point of the organ, especially on the surface, in the neighborhood of the ligaments, particularly on both sides of the suspensory ligament, as well as between its folds and in the tissue of the hepatic capsule and along the large portal veins. Of the lobes, the right one is perhaps the favorite seat of the process, but, generally speaking, syphilomata occur with equal frequency on the convex and concave aspects of the organ. In going through the literature it will be found that the greater number of luetic tumors

Multiple gummata of liver, showing marked scar formation about the gumma. Cross-section through left lobe.

arise in the right lobe of the liver, as has been recorded by Frölich, Helferich, Hochenegg, Legueu, Lilienthal, Roberts, Spencer, Trinkler, Wagner, and myself. Growths found in the left lobe of the liver have been recorded by Naumann, Mikulicz, Przewalski, Schmidt, Wagner, and myself. In the cases recorded by Bastianelli, Wagner, and myself the growths arose in the lower surface of the liver. In Helferich and Lauenstein's cases it occurred in the lobulus Spigeli. In Thompson's case the tumor arose between the right and left hepatic lobes below the suspensory ligament, while in Abbe's case the syphilomata were distributed over entire surface of organ.

Syphilomata have a great tendency to undergo fibrous changes, especially around their borders, where a tough fibrous capsule is frequently formed (Plate I). From the latter, fibrous strands and laminæ radiate into the surrounding hepatic parenchyma, giving rise to deep sulci and depressions produced by cicatrization, between which the hepatic parenchyma appears thick and uneven, giving rise to the so-called hepar lobatum. If these cicatricial strands are very extensive, large portions of the organ may become, so to speak, divided off from the rest. When this occurs, that portion which has become separated from the organ, according to the changes and its size, may become fairly movable, resembling a liver injured from too tight lacing. It may give rise to severe symptoms, while mechanically it may be the cause of ascites and icterus.

According to some authorities, ascites is believed to be a constant symptom of hepatic gummata, but, all things taken into consideration, this does not appear to be very frequent. In Spencer's and Wagner's cases there was very marked ascites; Spencer's case was aspirated several times, but the fluid rapidly collected. In one of my own cases, there was slight ascites, while in another case some ascitic fluid was removed during the operation. Spencer's case and two of my own had icterus. Icterus was also present in the cases reported by Helferich, Legueu, and Przewalski. In some cases the absence of jaundice is expressly mentioned. Generally speaking, it is never very marked; it results either from compression of the larger biliary passages, or from perihepatitis. When due to these factors its duration is not generally very long. However, if it results from an interstitial hepatitis developing simultaneously it will last for some time, or may even be permanent.

Single gummatous nodules are almost spherical, and, if they are not seated very deeply in the liver, they project more or less above the surface. When developing in the periphery of the organ they may be irregular in shape, on account of the fibrous tissue bands which radiate from them. The volume of hepatic syphilomata varies considerably, having been observed from the size of a millet seed to a hens' egg, or even larger, while as many as 50 have been met with and in some few instances a larger number has even been found. In one case the left lobe contained a number of gummata, varying in size from an almond to a walnut. In another case there was only one syphiloma, the size of an orange. Mikulicz removed a luetic tumor the size of a fist from the left lobe of the liver, while Bastianelli's syphiloma was twice the size of this and weighed 750 grams. In Schmidt's case the growth was as large as a small fist, measuring 8 cm. long, 6 cm. wide and 4¼ cm. in thickness. In Wagner's case the gumma was equal in size to three fists and weighed about three-fifths of the entire organ; the case observed by Hochenegg presented a rounded gumma, 8 cm. in diameter.

The consistency of these tertiary hepatic manifestations has usually been described as being hard and tough, as in the cases reported by Helferich, Legueu, Schmidt, Spencer, Thompson, Tricomi, Ullmann, and myself. In Lilienthal's case the growth was doughy, while in the one observed by Przewalski, it is described as soft and semifluid.

The surface of the growth was smooth and regular in Legueu's and Tricomi's cases; in Hochenegg's case it was fairly smooth and covered with a thin layer of hepatic parenchyma. In Schmidt's case the surface of the neoplasm was covered with depressed whitish areas. In the majority of the other cases it was irregular and uneven, particularly in those recorded by Helferich, Lucas, Spencer, Wagner, Thompson, Ullmann, Schmidt, and myself.

Much has been said about the color of these growths. They are described as being dark red, bluish red, dark blue, gray blue, light yellow, yellowish white, speckled white, cloudy, and markedly white.

The mobility of these growths naturally depends upon whether or not they are separated to any extent by deep sulci from the liver. If they are so separated they form a pedunculated growth separating

the neoplasm from the liver to a certain extent. That such a condition of affairs is not so very rare is shown from the fact that instances of this description have been recorded by Lauenstein, Legueu, Naumann, Schmidt, Tricomi, Wagner, and myself. In two cases the growth appeared as a tongue-like process, that recorded by Roberts reaching almost to the iliac crest. The second one reported by Trinkler extended towards the kidney. In cases of pedunculated growths, intestinal symptoms may arise from compression, leading to an erroneous diagnosis. The growth may be very movable, as in Wagner's case, where it could be pushed around in the abdominal cavity; but this physical condition will vary greatly according to the situation and shape of the growth. It usually can be moved from right to left as well as posteriorly, rarely upwards; very exceptionally, as in Thompson's case and my own, the tumor may be pushed downwards. Lauenstein calls attention to the fact that whenever the lobes of the liver are very movable syphilitic infection should be considered. It goes without saying that if there is any marked development of adhesions between the tumor and the surrounding structures, the mobility of the former must of necessity be limited, especially so, if the surrounding viscera are pretty well fixed in their position, but in Wagner's case the growth was very movable, although adhesions had occurred between it and the large intestine. Often, however, no adhesions are present, and in two of my cases, the growth was only adherent to the peritoneum. In Spencer's first case fibrous adhesions occurred between the tumor and the abdominal walls; in Frölich's case the neoplasm adhered to the rectus muscle, while in the first case recorded by Helferich it adhered to the omentum and surrounding tissues, while in his second case the tumor was bound down to the small curvature of the stomach and pylorus, as well as to the gall-bladder and duodenum; in Wagner's case it was adherent to the large intestine, likewise in Tricomi's. The liver is quite frequently more or less adherent to the diaphragm.

The patients will usually say that they have noticed the presence of the tumor for several years before seeking medical advice, partly because the growth gives rise to no symptom, and also for the reason that the pains are not particularly localized. Not infrequently there are no symptoms at all, or the patient may merely complain of a feeling of weight or pressure in the region of the liver. When pain

exists, it is usually felt in the right hypochondrium and occasionally radiates towards the back and shoulder. When pain is severe it is more apt to occur in periods varying from a few days to several months, while in the meantime the patient feels perfectly well. Pain may be produced by certain positions of the body, such as stooping, or a sudden backward motion, coughing, deep respiration, etc. Sometimes the patient complains of an increase of the pain at night. In those cases where an adhesive perihepatitis exists, the pain may become unbearable. Kehr says that "there usually seems to be no symptoms at the time the patient notices an increase in size at the upper part of the abdomen, but the pains apparently occur when the gummatous portions of the liver become divided off and, like a movable kidney, produce symptoms characteristic of wandering abdominal organs."

Anorexia, as well as nausea and vomiting, seem to be quite constant symptoms. In some cases constipation may be present, the fæces containing blood constituents. Later on, the constipation generally gives place to marked diarrhœa. Occasionally there is difficulty in micturition, attacks of vertigo, headache, and pains in the stomach. A rise in temperature and night-sweats are also met with, quite similar to those occurring in tuberculosis.

An extreme weakness with loss of weight and cachexia are almost constant, and in one of my cases the patient lost 30 pounds in eighteen months. Kaposi says: "The more complications (tuberculosis, scrofula, rickets, hæmorrhagic diathesis) occurring with syphilis, the more extensive will be the organs attacked by the luetic infection, which are physiologically necessary for nutrition and the circulation of important organs (liver, spleen, stomach, intestine, and the mesenteric and lymphatic glands), while the longer the syphilitic process exists, in many cases of the acquired and more frequently in congenital lues, a condition of general weakness and physical deprivation occurs, called syphilitic cachexia." This will be still more marked if amyloid degeneration of the viscera occurs.

Percussion over the tumor and the liver gives dulness when the syphiloma is located in this organ. If there is a detached tongue of liver substance containing the growth, and especially if inflation of the intestine is done, a line of tympanitic resonance is not infrequently elicited between the tumor and the liver, while at the same

time the tumor may be displaced, all of which contributes to the making of an erroneous diagnosis.

Examination usually reveals a protrusion in the right hypochondrium and in the epigastrium, while the tumefaction may even extend downwards far below the umbilicus. By deep respiration one frequently remarks that the liver and tumor follow the respiratory movements, but this, of course, is only possible when the tumor develops in the liver, or is connected to the organ by a short pedicle, or when no very marked adhesions are present. If the latter are present, it at once becomes evident why the patient complains of severe pain, which is only relieved by breaking up the adhesions. If the abdominal walls are thin or flabby, palpation will easily show the shape, consistency, and the surface of the tumor, as well as the degree of mobility and the existence of adhesions. The amount of tenderness on pressure also varies. In some cases there is none, while in others it is slight and can only be produced by deep palpation, while in very rare instances it may be severe, as in the case recorded by Ullmann. In the majority of cases the tumor is included in the hepatic parenchyma, but this is not always so, as the following case, which was under my observation some years ago, proves: A well-built man, forty years of age, had complained for several months of pain in the region of the gall-bladder, which varied in intensity, since which time he developed icterus. Temperature and pulse normal. In the region of the gall-bladder a large irregular mass could be made out. The border of the liver reached about three fingers' breadth below the costal margin. A diagnosis of hepatic tumor or some affection of the gall-bladder was made. On opening the abdomen some ascitic fluid escaped. The liver was enlarged, but in the region of the gall-bladder, where the tumor had been felt, only a mass of tough adhesions was found. These were broken down and revealed a normal gall-bladder. A diagnosis of duodenal ulcer was made, but nothing further was attempted, on account of the excessive adhesions. The patient's physician, who was present at the operation, made the remark that he had formerly treated him for syphilis, so that specific treatment was at once started and in three months' time all symptoms disappeared, and more than two years after the operation the patient was still in perfect health.

It is evident that all these various symptoms may occur, not only

in hepatic syphilis, but also in other diseases of the liver. It is therefore necessary to obtain an exact history and carry out a careful examination in order to discover former evidence of luetic infection.

Now, as many years may elapse since the initial infection before any specific infiltration occurs in the liver, it naturally follows that one rarely obtains correct information by questioning the patient. In four of the cases mentioned above syphilis was admitted, while in five it was suspected by the attending physician. If syphilitic infection is admitted, but no symptoms of lues are to be found, or rather, if there is merely a pathological process in the liver, then naturally nothing can be gathered under the circumstances for diagnosis. If the liver is evidently enlarged, with notched borders, if the surface of the tumor appears by palpation to be irregular, or if ascites or icterus is present, then in all probability one is dealing with a syphilitic process, especially so, if the patient is of that age when carcinoma is not apt to occur. However, even then one can only make a probable diagnosis. In this case, either mercury and the iodides should be immediately given in large doses, or perhaps, what is better still, an exploratory incision should be advised, for this alone can throw light upon the case immediately.

Syphilomata of the liver are much more obstinate to specific treatment than the same process in other viscera, a fact which I believe has been repeatedly proven, because in four instances already mentioned, an antisyphilitic treatment was tried without any result. Lennhoff relates a case where in spite of mercurial inunctions and the administration of the iodides, a semi-circular, hard, and irregular tumor of the liver about the size of an orange was in no way influenced by the treatment, and what is more, it in no way prevented the development of a second gumma. At the autopsy, the patient having died of nephritis, both the tumors were at once recognized as syphilomata. On the other hand, very favorable results have been reported accruing from antisyphilitic treatment; thus, for example, Leichtenstern has published a case of a male forty-eight years of age, who had a large tumor of the liver, accompanied by ascites and extreme cachexia, which was thought to be carcinoma for a considerable length of time, but the process was completely cured by mercurial inunctions. Ziemssen encountered a very extensive syphilitic tumor of the liver, which considerably diminished under energetic

methods, as in the case of the patient whose tumor extended down as far as the pubis. In another instance the specific neoplasm reached the umbilicus, and after treatment the liver regained almost its normal size, the organ only extending about one finger's breadth below the costal margin. Basing himself on these results, the latter mentioned authority has advised employing an energetic specific treatment before resorting to surgical interference.

On account of the obstinacy of hepatic syphilomata, it is absolutely necessary to employ very large doses of the iodides and mercury, while the treatment should extend over a very long period of time. In Trinkler's case the tumor disappeared completely by a constant and energetic antisyphilitic treatment, although the liver remained somewhat enlarged; specific treatment carried out for thirty days prior to the operation had been unsuccessful. It is most peculiar to note that in just the cases where there appeared to be a syphilitic history and in which suspicions of a specific hepatic process was justified, it proved to be a fallacy, whereas in those cases where the history revealed nothing and where there were no apparent symptoms of lues, where all kinds of diagnoses had been made, the tumors after removal proved to be syphilomata. In some cases, where the patients only experienced the inconveniences of an antisyphilitic treatment, no harm accrued, but these diagnostic errors have also been fatal to the patients. Lennhoff has reported the case of a patient fifty-two years of age, who had had a specific infection, followed by several relapses. For years he had been the possessor of an hepatic tumor. The liver was hard, and on its surface were several irregular, rounded nodules, which appeared to have a small depression in their centre. The diagnosis of syphilis was taken for granted, and, consequently, antisyphilitic treatment was started, without any result. A little later, by rather rough palpation a distinct crackling was both heard and felt and the supposed syphilomata proved to be a hardened liver containing echinococci. Another case was that of a male forty-eight years of age, and here, too, the liver was quite hard and extended almost to the umbilicus. Over the surface of the organ three uneven and irregular elevations were made out, possessing a somewhat softer consistency than in the preceding case; one of these projections had a depression in its centre. This process was of three years' standing and was at first

thought to be carcinoma of the liver. The patient only complained of a sensation of pressure in the hepatic region. Twenty years previously he had been infected and had had several recurrences, and, on account of this, the diagnosis of hepatic syphilis was made and inunctions and iodides ordered. The treatment was continued without any improvement for six weeks, but the diagnosis was not revised as this is not at all unusual in hepatic syphilomata. However, upon a renewed examination, a deeply seated fluctuation could be indistinctly felt, and upon puncture a watery fluid was withdrawn, which, upon microscopic examination, was found to contain hooklets of the echinococcus.

Referring to these cases, Lennhoff points out that for differential diagnosis neither the semicircular shape nor the hardness of the tumors is characteristic of syphilomata, and also that a depression in the centre of the tumor may occur in cases of echinococcus infection. Another case is reported by Puritz, where an error in diagnosis proved fatal to the patient. He was a man twenty-nine years of age, who had developed an ulcer on the penis in 1885, which was thought to be specific in nature. As a precaution, the patient was put upon antisyphilitic treatment. There were no secondary manifestations. In the fall of 1889 he noticed that the right side of the abdomen was increasing in size, but the physicians who attended him became assured that the tumefaction on the right side was due to an enlargement of the liver, which was considered specific, and the patient was immediately placed upon antisyphilitic treatment, which, however, was unsuccessful. In February, 1890, there was a rise in temperature, while in May of the same year the diagnosis of malignant neoplasm of the liver was made. In August, the patient entered the clinic of Prof. Tschudnowsky, and here five hard nodules the size of a fist were discovered, which could not be felt through the tense abdominal walls. No connection could be found between the liver and the tumors. The axillary and cervical glands were enlarged and some of them felt hard. From these facts, and particularly on account of the irregular rise in temperature, which could be accounted for by nothing else, the diagnosis of sarcoma of some abdominal organ was made, probably of the mesentery and the retroperitoneal glands. The patient died from exhaustion in about two months' time, and autopsy revealed primary sarcoma of

the liver. Whether the patient might have been saved by a primary operation is doubtful, but, at any rate, an exploratory incision would have immediately cleared up the case. Langenbuch, Tricomi, and myself have met with similar cases.

If there is extreme cachexia, which, as has already been mentioned, is not so very infrequent, and if all evidences of syphilis are wanting, a syphiloma of the liver can easily be mistaken for a malignant neoplasm. Since carcinoma and syphilis often occur together, it consequently happens that in these cases where distinct specific symptoms are present, such as papules, exostoses, cutaneous gummata, etc., while the tumor increases rapidly in size and the patient is at the age where carcinoma is apt to occur, the result of a specific treatment should not be awaited, and an explanatory operation should at once be resorted to.

In my paper published in the *Archiv fur klinische Chirurgie,* I made the statement that " the rapid development of gummata of the liver very frequently resembles carcinoma." We have neither any distinct systems, nor methods of examination which will aid in any way in instances where it is doubtful whether or not a syphiloma of the liver is present. When there is a syphilitic history, or when luetic symptoms are present, we then can assume with all probability that the process is of syphilitic nature. If the tumor gives evidence of slow growth, the symptoms mild and insignificant with no emaciation or cachexia, a specific treatment of mercury and iodide combined in very large doses should be essayed, but I believe that in all cases an exploratory incision is decidedly indicated. When the abdomen is opened, whether the previous diagnosis is correct or incorrect, and a gummatous process is found to exist, the question then arises whether the disease should be extirpated or not. Generally speaking, only a small proportion of cases of syphiloma of the liver is susceptible for operative measures; namely, (1) a lobe of the liver which has undergone syphilitic degeneration and has become pedunculated, and (2) encapsulated syphilomata. The first are more readily removed by operative interference, because the application of a tight ligature around the pedicle will control all hæmorrhage. The second type of process can be shelled out of the capsule, as many operators have done. If there is a large gumma on the border of the organ, easy of access, and if medical treatment

should be unsuccessful on account of the extensive alteration of the tissues, extirpation would then be justifiable, provided that the hæmorrhage can be controlled, that is to say, if there exist cicatricial contractions around the process, which, on account of their compression on the vessels, would naturally lessen the extent of the bleeding. A syphiloma, when seated deep down in the midst of the hepatic parenchyma or on the under surface of the liver, cannot be removed. If there are several gummata, or many tough adhesions rendering the operation very difficult or even impossible, their removal should not be attempted. If there is very marked ascites, it should be recalled that from the mere fact of the removal of the syphiloma, the ascites may not of necessity disappear, and it is rather more probable that the process of contraction resulting from a pedunculated gumma has in all probability resulted in a constriction of the hepatic veins, than that strands of connective tissue, which radiate into the hepatic parenchyma, produce compression of the vessels resulting in stasis and finally ascites. In such cases no relief will be obtained by extirpation nor can inunctions and iodides relieve this mechanical disturbance, for the simple fact that bands of cicatricial tissue can be hardly, if at all, influenced by specific treatment. Langenbuch very justly remarks that in the first place the diagnosis should, if necessary, be verified by an exploratory incision, and operation should only be undertaken if one is certain of bringing immediate relief otherwise, the abdominal incision should be closed and inunctions immediately started. In 32 cases of hepatic syphiloma, including my own, which were operated upon after a correct or incorrect diagnosis had been made, the true character of the tumor was apparent after the abdomen had been opened, while in others a correct diagnosis was made only after removal of the growth. In 10 cases no diagnosis was made, in 3 the diagnosis of hepatic tumor was arrived at, 5 times the cases were supposed to be malignant growths of the liver, 1 was supposed to be an abdominal neoplasm arising in the kidney or the stomach, or a perigastritis, pyloric neoplasm, neoplasm of the liver, deep-seated abscess of the liver, malignant tumor of the liver or kidney, or tuberculosis of the transverse colon, syphiloma or echinococcus infection of the liver, syphiloma or adenoma of the liver, syphiloma or carcinoma of the gall-bladder, perhaps syphiloma or purulent cholecystitis, or some affection of the liver.

GENERAL ANÆSTHESIA UNJUSTIFIABLE IN THE RADICAL CURE OF INGUINAL HERNIA; LOCAL ANÆSTHESIA IN MAJOR OPERATIONS*

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GENTLEMEN: It may seem somewhat radical to assert, since so many surgeons use general anæsthesia in performing operation for the radical cure of inguinal hernia, that this procedure is unjustifiable; but I think that I am in a position to make good this statement, since in the last five years I have had an experience of 428 operations for inguinal hernia in about 400 patients, and all of them have been done with local anæsthesia. No matter how carefully general anæsthesia may be secured, there is always an element of danger in it, and besides patients are discouraged by dread of it from having their inguinal hernias treated by an operation for their radical cure.

With regard to the danger in the operation, I think that the best proof of its complete absence is to be found in the fact, that not only were there no deaths in this series, but in not a single case did suppuration occur. This latter claim will doubtless seem to many exaggerated, since in such a long series of cases some defect of asepsis would surely occur to produce at least superficial infection. As a matter of fact, however, the local anæsthesia has been of service also in saving the patients from danger in this regard. Infection is due, not only to the presence of infective agents, but also to the lack, at a particular moment, of resistive vitality in the tissues. The more tissues are handled and roughly treated during the course of an operation, the more danger there is of subsequent suppuration. Inasmuch as the patient is in full possession of his senses and not infrequently continues to laugh and joke with the surgeon during

* A clinical lecture delivered at the New York Polyclinic Hospital and School for the Graduates in Medicine.

the entire operation, the use of local anæsthesia constantly keeps in the operator's mind the necessity for the most delicate manipulation of the tissues. The full consciousness of the patient does not prevent the operator from following out all the steps of the operation; but it keeps him constantly mindful of even the slightest possible pain that might be unnecessarily inflicted on the patient.

Fifteen of the cases which I have mentioned as occurring in my service, were strangulated hernias. These were operated upon quite as successfully and with just as little pain to the patient as those in which the time for operation was left to the patient's choice, and in which he did not come to the table in the state of excitement and worry so apt to be associated with a strangulated hernia. This question of strangulation, however, brings us to the crux of the hernia question. A man who carries around an inguinal hernia has constantly a potential cause of death with him. If the hernia is well retained by a truss, the danger may not amount to much; but even in these cases it is always present, and the truss itself may, after some unusual effort, prove the immediate cause of the strangulation.

There is, however, another and even more important aspect to this question. The economic side of hernia is not often discussed, yet it is a feature deserving very careful attention. Professor Graser, in Von Bergmann's System of Surgery, recently translated into English under the editorship of Professor Bull states that of the whole population about 1 in 20 to 1 in 30 suffers from rupture. There are many good authorities who give the number of hernia patients among the population as even higher than this, some stating that it is as high as 1 in 15. Four-fifths of these cases are inguinal hernias. The vast majority of them occur among wage-earners. The capacity of the wage-earner to perform his daily labor is greatly reduced by the fact that he has a hernia. According to the German system, by which the government, in return for certain sums paid to it by the laborers and their employers, guarantees to take care of them when they become disabled, it is understood that a man suffering from hernia loses about 15 per cent. of his ability to earn wages if his hernia can be completely retained by means of a truss. If this is not the case, however, his disability is considered as much higher, even up to 50 per cent.

Another word with regard to the economy of trusses seems advisable. Trusses never cure hernia except in the cases of children under four years of age. When a man once begins to wear a truss, he will have to wear it, as a rule, for the rest of his life. Trusses cost, even for the poor, from $5 to $15; and with the best of care their average life is not more than from three to five years. It has been calculated that the ordinary individual will have to buy at least six, and usually more, trusses during his life-time. A little calculation will show that this expense for trusses would pay the ordinary surgeon's fee for an operation for the radical cure of the hernia. After the radical cure the patient has not the bother and discomfort of wearing a truss; he is not in the danger of having his hernia strangulated; and, besides, he is able to do more work and to earn better wages. There is every reason, then, to simplify the operation for the radical cure of inguinal hernia so that everyone may be more willing to have it performed.

If we remember that there are probably 2,000,000 wage-earners in this country who are sufferers from hernia, most of them from inguinal hernia, the great need of simple, easy, ready methods of operating on such hernias is at once evident. The methods must be such also as will not deter a patient from having the operation done. The removal of the necessity for general anæsthesia is extremely important in this matter, and men can readily be persuaded that the operation is not serious if it can be done without their being put to sleep.

I may say at once that in none of my cases was there pain enough to cause the patient to flinch or jerk even involuntarily. Most of my operations have been done on very intelligent persons, and many of my patients were physicians. One of them once told me, that beyond the sense of uneasiness that something was going to happen, there was no more feeling of discomfort than if he were being shaved or were having his hair cut. As I have said, many continued to talk pleasantly with me all during the operation.

I am not ready as yet to report the recurrences from these operations, because I am still engaged in the difficult task of collecting statistics. I am sure, however, that no more occur than in the experience of others, and I think less.

The method of operation by the use of local anæsthesia necessi-

tates the careful selection for purposes of anæsthesia of those nerve-trunks which supply that part of the abdomen involved in the operation. As the result, the nerves are not divided, as is sometimes done when the operation is performed under general anæsthesia. The division of these nerve-trunks leads to atrophy and flabbiness of the muscles of this region, and consequently predisposes to a return of the hernia. It is well known, for instance, that when the ilio-inguinal nerve is divided in the operation for varicocele the cremaster muscle relaxes and leads to a dropping of the testicle and a dragging down of the scrotum. It seems clear that this same thing should hold for the internal oblique, which is supplied by the same nerve. No text-book emphasizes the necessity for the exercise of care in the preservation of this nerve; but from analogy it may be readily understood how the muscles of the abdominal wall may relax as a consequence of the division of their most important nerve supply. It was Dr. Cushing, of Johns Hopkins, who first pointed out the course of the nerves in the neighborhood of the incision for the radical cure of inguinal hernia, and who first suggested the advisability of allowing them to remain intact, since their severance would lead to atrophy and flabbiness of the muscles and so predispose to the recurrence of the hernia. From this suggestion came the method that I will describe of cocainizing these nerves, and thus securing complete nerve block all over the field of operation.

The ease with which the operation for the radical cure of inguinal hernia can be done will be best appreciated if we consider the restricted region of the field of operation, and the superficial position of the whole area in which the operative manipulations are undertaken. As I have said, there is neither nerve-trunk of importance nor any vessel of considerable size encountered in the skin or subcutaneous fascia. Beneath the aponeurosis of the external oblique lie the three nerve-trunks which preside over sensation in the entire field of operative work: the inguinal branch of the ilio-inguinal, the hypogastric division of the iliohypogastric, and the genital branch of the genitocrural. If all three are found and cocainized the operation is totally devoid of pain.

Their identification is not difficult. The first two lie between the two oblique muscles directly under the incision. The genitocrural is usually among the structures of the cord. I have spoken of

the nerves in the order of their importance as pain producers, and in the order of their constancy in anatomical position.

All three are derived from the first and second lumbar trunks, and participate feebly in forming the lumbar plexus before they proceed to their destinations over the field of this operation. Intercommunicating branches pass from one to the other throughout their course, and this explains the comparative freedom from pain after cocainization of the ilio-inguinal, even though the other two have not been found. The largest, most constant, and easiest found is the ilio-inguinal. It runs from the iliac crest inwards, resting upon the surface of the internal oblique, and is always to be found running a course parallel to the long axis of the hernial protrusion, sometimes as far outwards as the shelving process of Poupart's ligament. If the ilio-inguinal is large, the iliohypogastric is small and inconsequent, so that little time should be spent in search for it. In fact, the iliohypogastric may be ignored if the margin of the internal ring is properly infiltrated. At times it may be absent, but when present it generally lies upon the internal oblique an inch or more above the arching fibres of the internal ring. The genitocrural is also inconstant in situation, and is frequently not identified during the operation. It emerges from the internal ring, along the cord, and consequently behind the neck of the hernial sac. As a sensory nerve it is of little consequence.

The method of local anæsthesia consists essentially in cocainizing these sensory nerve-trunks and thus in accordance with Corning's discovery abolishing all pain sensation throughout the area of their distribution. This discovery of "nerve blocking" makes possible the performance of painless major operations, even to the extent of abdominal sections or amputations of the leg and arm. Personally, I have performed three amputations at the thigh under simple local anæsthesia by this method with the assistance of massive infiltrating injections of one to ten thousand cocaine, such as were suggested originally by Matas, of New Orleans. Usually local anæsthesia is selected in such operations because of the danger of general narcosis, from either chloroform or ether.

Altogether during the operation for inguinal hernia not more than one-half grain of cocaine is used. In all of my 400 cases I have never had to employ more than one grain of cocaine, and in less

than a dozen cases more than one-half grain. This amount of cocaine is not only not dangerous to a healthy patient, but seems actually beneficial to one suffering from shock. In severe accidents, where I have had to amputate at the hip and the patient has been in severe shock at the beginning of the operation, there was not only no increase of this condition, but the patient was actually better at the end of the operation than at the beginning. This bears out the statement of Crile and Matas, that injection of the sensory nerve-trunks prior to their division entirely prevents shock. Certainly in my cases I have found this to be true. It seems, indeed, to be a new acquisition to our knowledge of the pathology and therapeutics of shock.

My own technic for local anæsthesia is as follows: 1 grain of cocaine is dissolved in an ounce of sterile salt solution, making a cocaine solution that is practically 1 to 500 in strength. This fluid is used to infiltrate the skin along the line of incision, and also to cocainize the nerve-trunks when they are found. For all subdermic infiltration a solution of 1 to 1000 is used. The second dilution is made by diluting, in the syringe, the stock solution with an equal amount of physiological salt solution. We have experimented with solutions stronger and found them unnecessary, while weaker solutions do not produce perfect anæsthesia. When the tissues are properly infiltrated, a solution of 1–500 strength will completely anæsthetize the skin for one hour and a half.

It should be made fresh for each operation, as in cocaine solutions a fungus rapidly develops. In a few hours it is potentially septic, and in a few days will produce suppuration. It is conceivable that a solution of pure water might interfere with healing, because of its tendency to swell the tissues by imbibition; but when the law of osmosis is observed and physiological salt solution is used, a solution isotonic with the blood serum, it cannot interfere with the healing of the wound. A cocaine solution may be boiled long enough for sterilization without detriment to its anæsthetic value, but if boiled too long the drug is decomposed and becomes dangerous. As a matter of fact, the blame for many complications and sequelæ during and after the operations done by local anæsthesia has been placed quite unjustifiably on the cocaine employed. There is a tradition, for instance, accepted by many surgeons that cocaine

wounds do not do well. This is entirely due to the fact that stock solutions of cocaine were employed, in which fungi had developed, and which were thus responsible for the failure of union by first intention. My own experience with freshly prepared cocaine solution, 400 patients having been operated upon without suppuration, proves that cocaine does not predispose to wound infection.

In the same way, the symptoms of supposed cocaine poisoning when analyzed prove nearly always either to be psychic, following nervous excitement, or be due to an idiosyncrasy of the patient for cocaine. It is interesting to remark, however, that, in literature, probably all the cases of cocaine poisoning, and especially those ending fatally, were published more than a decade ago, when the proper methods of obtaining thorough local anæsthesia by means of cocaine had not been elaborated. What we would now consider very strong solutions of cocaine were employed in those early days. A 4 per cent. solution, for instance, was suggested at first and only slight dilutions were supposed to be made. This could scarcely help but produce symptoms, since considerable quantities of cocaine, 2 grains and more, were occasionally thrown into the system. A strong solution of cocaine will not produce any better anæsthesia than a weak one. A certain amount is needed, but since it is absorbed, all employed beyond this point is not only useless but harmful. It seems necessary to dwell on these points in order to encourage some of those who doubt the advisability of using cocaine as I have suggested.

Many of the idiosyncratic effects reported were psychical phenomena and were not due to the drug. This psychical shock is occasionally noted now in our work, but it is properly understood. The symptoms are in part similar to those of cocaine poisoning—pallor, sighing, rapid pulse, and facial perspiration. Lowering the head below the level of the body and hypodermic injection of camphorated oil, will bring about rapid restoration, and the operation proceeds with the additional use of any cocaine needed to control pain.

The maximum safe dose of cocaine in all cases, under all circumstances, has not been determined. Analogy suggests that there is a safe dose, as in the case of strychnine, hydrocyanic acid, and other poisons; and none will gainsay that intermittent injections throughout the period of an hour, amounting to two-thirds of a grain, are well within the limits of absolute safety.

Morphine is the true antidote for cocaine, and a solution, properly prepared, should always be at hand in case genuine symptoms of cocaine poisoning should occur. Personally, I consider that morphine also acts as a prophylactic against many of the unpleasant consequences of cocaine, and consequently I always give patients a quarter of a grain of morphine hypodermically about half an hour before the operation is begun. Without making them sleepy, to any extent, this allays most of the excitement incident to the operation, and seems to prevent unpleasant effects from the cocaine. I prefer to use morphine before the operation rather than to follow Schleich's method of dissolving the morphine with the cocaine and using the composite solution. The morphine does not act as rapidly upon the individual as the cocaine, and therefore is better introduced beforehand.

The interesting question always remains why certain operators can use local anæsthesia with good success while others cannot. It is evidently due to a difference of personality. Certain surgeons, though excellent operators, are always at high tension during the course of an operation, and their nervousness is inevitably communicated to the patient, with the result that even slight discomfort is exaggerated into almost unbearable pain. The greatest care must be exercised not to disturb the patient's mind. A sign is quite sufficient to indicate to the properly qualified assistant that the moment for the incision has come. If a knife must be asked for the use of the word bistoury will often be sufficient to conceal from the attention of the anxious patient that the actual cutting is about to begin.

It must be distinctly understood, when I talk so strongly of the advisability of using cocaine for hernia operations, that I mean inguinal hernia and not hernia without distinction. The average physician seems to think that every form of hernia can be operated upon in the same way. This is not true, however, with regard to femoral and ventral herniæ. We cannot secure so readily the nerve-trunks supplying the sensory nerves to the affected areas, and consequently the local anæsthesia is not so complete.

One of the most serious sequelæ to general anæsthesia is the nausea and vomiting. This difficulty is entirely obviated by local anæsthesia; and there is no doubt that it is an important gain in the operation. Sometimes the very severe nausea and vomiting may

even produce serious effects upon the field of operation. Powerful abdominal muscles have been united, and in their contraction during vomiting they may very well tear out at least some of the deep sutures, thus spoiling the effect of the operation. There seems to be no doubt in the minds of many that the success of the operation for the radical cure of hernia has been seriously impaired because of this violent straining during vomiting.

On the other hand, it must not be forgotten what the effect of such disturbance is upon the minds of patients themselves, and upon their friends who come to see them. After having witnessed the extreme sickness to which long-continued general anæsthesia may subject a friend, no one, who is himself a sufferer from hernia, can be so readily persuaded to have the operation done. The patient himself, with the memory of this, is not apt to encourage others in the matter of operation; and as a consequence many persons continue to bear the discomfort and inconvenience of trusses with the danger of possible strangulation of their herniæ, on account of this unfortunate discouragement. All of these discomforts are entirely done away with by the use of a local anæsthetic which, at the time of operation, does not in the least add to the patient's discomforts.

There are no contraindications to the use of local anæsthesia for the radical cure of hernia. The size of the hernial protrusion, as I have said before, does not constitute a limitation to the employment of this method. I have operated on two patients where the hernial tumor was the size of a child's head, yet the operation in each case was quite as free from pain as any of the others in my list. When the patient is very fat, this suggests limits to the use of local anæsthesia more than does the size of the protrusion. In these cases the wide retraction of the tissues necessary to expose the field requires a longer incision. The thick adipose layer prevents infiltration anæsthesia from proving effective, and more of the solution must be applied. Even with a considerable amount of fat, however, local anæsthesia can be absolutely effective. Neither are there any complications which contraindicate the performance of the operation with only local anæsthesia. I have found the appendix as part of the contents of the hernial sack five times, and in each instance it was amputated and the stump inverted without additional cocaine, and without pain. Excision of existing varicocele adds nothing to

the pain, and I have amputated a large mass of omentum weighing sixteen ounces, with only the production of some shock and nausea, but no pain. The cutting and tying of the omental blood-vessels does not produce pain, though it must not be forgotten that blood-vessels elsewhere are always accompanied by little nerves that give rise to pain on manipulation. It must be remembered, also, that traction on the omentum always produces nausea in the patient. Forcible traction on the omentum will cause both pain and nausea. This is not necessary, however, in the operation for the radical cure of hernia, and the necessity for care in manipulation actually does good by making the surgeon more gentle.

ASEPSIS AND ANTISEPSIS

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The wonderful achievements which characterize modern surgery are chiefly due to two things,—the discovery of anesthetics and the establishment of the fundamental precepts of surgical cleanliness. The latter were first laid down by Sir Joseph Lister in 1867–68, but even before his time, prominence was accorded to various antiseptic measures. Good results were reported from the use of carbolic acid some seven years previously by Küchenmeister of Dresden; Lemaire's work on carbolic acid was published in 1863, while three years later, Campbell de Morgan called attention to the use of chlorid of zinc as an antiseptic. Lister, basing his theories on Pasteur's observations on the action of bacteria in producing fermentation, proposed certain measures for preventing the entrance of microörganisms into the wound. His plan was founded on the belief that the infection of wounds occurred from contact with the air which contained the germs or their spores, and he therefore directed his efforts to the destruction of this low order of vegetable life before it gained access to the wound. This he sought to accomplish by saturating the air of the operating room with a spray of carbolic acid solution.

It is true that the air can be the medium of wound infection to a certain extent, for it has been demonstrated that dry air contains dust in which bacteria or their spores are present to a larger extent than in moist air. Then Koch came forward with the claim that most of these bacteria are innocuous, and that infection was more generally caused by germs being brought into direct contact with the wound by the clothing of the patient or from the skin itself.

It is well known that suppuration and wound infection depend to some extent on the relative number of bacteria which have entered the wound, their virulence and the resistance offered by the tissues and body fluids of the patient. The latter is expressed by the germicidal qualities of the blood serum, and even where the

infectious agents are already present in a wound, suppuration may thus be prevented. The latter process takes place, however, when the number or virulence of the bacteria are too great to be overcome in this manner. An explanation is thus afforded of the behavior of certain wounds which terminate favorably even where neglected or imperfectly treated.

In order to obtain the best results in the treatment of wounds it is essential that a thorough acquaintance be had with the fundamental principles of surgical bacteriology. These teach us that every portion of the human anatomy which is on the physiological exterior of the body affords lodgment for bacteria, which become septic agents as soon as this barrier is passed. Sepsis may be defined as the effects resulting from the entrance and multiplication of microörganisms and the absorption of their products in the body. The condition is characterized by local inflammation and constitutional symptoms which include fever, and disorders of the nervous and visceral systems. Clinically, suppuration in a wound may be looked upon as the invariable accompaniment of bacterial invasion and if these organisms are excluded it will not occur.

Methods of cleansing a wound or the unbroken skin previous to operation may be designated as aseptic, antiseptic, or a combination of the two. Asepsis aims at a thorough sterilization of the parts and of all materials and objects brought into contact with the wound, and exclusion later of germs from the wound by means of occlusive dressings only. Antisepsis, however, is that method which keeps germicidal agents constantly in contact with the wound after sterilization has once been secured. Antiseptic methods were formerly greatly in vogue, but in present day surgical practice they have largely been superseded by the aseptic method or the combination already referred to.

Wound complications, unless brought about by some inherent constitutional trait, are almost always due to the presence of a foreign body or to microörganisms. In order to prevent these, or to diminish their intensity when already present, it becomes necessary to employ means for their removal and destruction. These may be either mechanical, thermal, or chemical, but in every case it should be borne in mind that the tissues themselves are an important factor in preventing the spreading of an inflammation and

that they must be injured as little as possible by whatever procedure is employed.

Mechanical disinfection is limited in its application to the wound and the surrounding skin, but is also one of the best means for removing infectious materials from the surgeon's hands and instruments. Soap and hot water with sterilized brushes are the only implements necessary. A countless number of soaps have been introduced for this purpose, some of which have germicides or gritty materials added in order to facilitate the cleansing process. The best and also the most commonly adopted is the tincture of green soap (Linamentum Saponis Mollis) which consists of soft soap, 65 parts; alcohol, 30 parts; oil of lavender, 2 parts; and water to make 100 parts. When using the brush too much force must be avoided, as the skin is likely to be abraded by the bristles and thus exposed to the possibility of subsequent infection and interference with proper healing. In using the green soap it is essential first to rub it thoroughly into the skin and then apply enough water to form an abundant creamy lather. By this means bacteria in the follicles of the skin will be brought nearer to the surface by the bubbling of the soap and are more readily removed. This subject will be referred to again on another page.

Thermal disinfection is the most reliable and an easy means of destroying bacteria or other microörganisms. It can only be applied, however, to instruments and dressings. In wounds it may be employed to destroy localized sloughs or other infectious agents, in the form of the actual cautery. Boiling water, steam and dry heat are the agents usually employed. The efficacy of boiling water in destroying bacteria is shown by the fact that certain bacterial spores which are killed only by a continuous soaking for a week in germicidal solutions, can be destroyed by boiling water in a few minutes.

Chemical substances formed the basis of all the early methods of sterilization but have now been assigned a single rôle in the process. Among the essential attributes of these substances it is necessary that they should be marked by penetrability, rapidity of effect, and a selective action on the offending microörganisms. These factors must not be sacrificed to any possible irritation or destruction of the tissues. Very few chemical substances meet

these requirements, and of the large number proposed only a comparatively small number have survived the ordeal to which they were subjected.

Of the two great procedures for the attainment of surgical cleanliness, the antiseptic method was the first to be employed. Here the sterilization of the wound, the field of operation, the hands of the surgeon, the instruments and all other materials coming in contact with the wound, is secured by the use of germicidal solutions. The latter are also used to irrigate the wound during an operation, and this completed they are applied in the form of a dressing. It is now admitted that this method has many disadvantages. Some germicidal solutions have been found to lack the powers attributed to them, and others are accompanied by an irritant action or enter into chemical combination with the tissues, thus destroying them or forming an insoluble coating under which bacteria may thrive and multiply. It is also known that these substances may cause the microörganisms themselves to be surrounded by an insoluble and impervious envelope, by which they are most effectually preserved. Then again, antiseptics applied to a wound may become absorbed and produce symptoms of intoxication in the patient.

When the aseptic method is employed, the field of operation and the surgeon's hands and instruments are sterilized by germicidal solutions or heat, and no further antiseptics are applied to either. For irrigating wounds, sterile water or normal salt solution is employed. The dressings are sterilized by heat and not treated with germicidal substances. They are kept as dry as possible, it having been proven that bacterial development may be thus inhibited. This method in contrast to the antiseptic is attended by numerous advantages. Among the most important of these is the fact that the vitality of the tissues is retained. In addition, it admits of ready application, causes little or no irritation, and toxic effects from the absorption of poisonous materials in the dressings are avoided.

The methods of sterilization, as applied to various localities, will now be briefly considered.

Sterilization of the surgeon's hands is one of the most important steps preliminary to an operation, for it has been shown that the

hands of the operator are one of the commonest sources of infection in operative procedures. The essential point demanded of an effective method is that repeated use will not be followed by any undue amount of irritation. Mechanical cleansing with soap and water has now come to be regarded as the *sine qua non* of all methods of hand sterilization and constitutes the first step in almost every one proposed. The hands, forearms and fingers of all who are to take part in the operation should be thoroughly scrubbed with green soap and a fairly stiff brush for a period of fifteen minutes, *by the clock*. It is well to use running hot water or at least to change the supply of water several times. The parts most likely to harbor germs are the interdigital folds and the recesses about the nails, and, therefore, particular attention should be paid to these localities. It is essential, moreover, that a medical man keep his nails well trimmed not only for appearances' sake, but also to render their cleansing an easier matter when preparing for an operation. The nail should be cut round so as to conform with the outline of the end of the finger and then smoothed with a file. The mistake is often made of clipping them too short, for this is apt to render the extremities of the fingers very tender and interferes with their strength and sense of touch. All shreds and hang nails should be carefully cut away with a pair of sharp-pointed scissors. During the process of washing, the dirt may be most readily removed from under the nails by the use of pointed sticks of orange wood provided for this purpose. Inflammation of the hair follicles sometimes occurs on the dorsum of the hands and the forearms when the general health is somewhat impaired and the resistance diminished. This condition may prove a source of infection to the patient, and, when present, the hair should be extracted from the infected follicle, and a drop of tincture of iodin applied to the spot. If the surgeon is afflicted with any infectious disease or localized infectious process, it would be the part of wisdom to decline to operate. The presence of a small wound of the finger or arm also necessitates precautionary measures when the surgeon finds it necessary to perform an operation. If any signs of inflammation are evident, the wound or abrasion can be disinfected with carbolic acid applied on the end of a probe or toothpick, followed by the application of a little alcohol, and then sealed with flexible collodion. This should be done after

the preliminary washing and then a sterilized rubber finger cot worn. After the hands are completely sterilized, the wearing of rubber gloves will not be considered necessary.

After scrubbing, the hands should be thoroughly rinsed in rapidly running water, or, if this is impracticable, at least in several changes of water. This effectually removes all material softened by the soap and loosened by the brush. Following a preliminary washing with soap and water, some surgeons rub the hands with rectified oil of turpentine in order to dissolve the fatty secretions of the skin, or, if this has been omitted, the hands may be rinsed in alcohol or ether after the soap and water scrubbing has been completed.

Immersion in some germicidal solution should follow, although some authorities claim that the hands are sufficiently sterile when the steps just enumerated are completed. A solution of bichlorid of mercury (1:1000) is commonly employed and in this the hands and arms should be rinsed for from three to five minutes. Where pus has been touched within a comparatively short period, it is recommended that the hands after scrubbing, be immersed in strong carbolic or lysol solution for a moment, after which they must be immediately rinsed off with strong alcohol in order to prevent the caustic effects of these substances.

Howard Kelly has suggested a method for sterilizing the hands which has proved very satisfactory but is apt to be irritating to sensitive skins. The hands and forearms are first scrubbed with soap and water in the manner already described and then dipped for a minute in a warm saturated solution of potassium permanganate. The resulting deep mahogany stain can be readily removed with a warm saturated solution of oxalic acid, which in turn may be washed away with sterile water. This method depends for its efficacy on the deoxydizing properties of the potassium salt.

Another procedure largely adopted is that suggested by Semmelweiss, in which the germicidal action of chlorin gas is relied upon to destroy the microörganisms present on the skin. After scrubbing the hands with soap and water, a tablespoonful of commercial chlorid of lime is taken in the hands together with a lump of carbonate of soda the size of a walnut, and enough sterile water to make a paste. This is rubbed over the hands and arms for

several minutes until the granules of lime disappear and the skin feels cool. This is followed by a rinsing in sterile water. A number of other procedures have been suggested, but those mentioned are the most generally employed.

Recognition of the unreliability of any one plan of hand disinfection led surgeons to seek a method by which the operator's hands could be enclosed in an envelope which could be rendered absolutely sterile through means which could not be applied to the human skin. Gloves were tried, made of various materials, but at present those manufactured in one piece without joints, of thin rubber, have largely superseded all others. The addition of the latter to the surgeon's armamentarium may be looked upon as the crowning effort in the development of surgical technic. Having eliminated the hands as a possible source of infection, the skin of the patient alone constitutes the doubtful factor in the healing of the operative wound. But since the introduction of the rubber glove, the results have been so uniformly good, that it may be safely stated that the skin of the patient is seldom the source of a wound infection if properly treated before operative procedures. The only objection that can be urged to the universal employment of rubber gloves is their expense, lack of durability and interference with the dexterity and sense of touch of the operator. The latter objections are soon overcome when one becomes accustomed to their use, and if properly taken care of, they should prove an economy, as their cost may be credited with the amount saved in such items as dressings and antiseptics which may be necessitated by an infected wound.

Rubber gloves should be cared for in the following manner: After an operation they must be thoroughly washed with soap, but every trace of the latter should then be removed by frequent rinsing in cold sterile water. In order to dry them thoroughly they must be turned inside out several times, the drying being accomplished with a towel or by hanging them in a current of air. They may be then kept until wanted again by dusting them all over with talcum powder and laying them between tissue paper or the folds of a towel. Rubber gloves are preferably kept in a tightly covered box which is long enough to hold them without folding, and stored in a dry, cool place. When purchasing, it is well to select a size

larger than that of the gloves ordinarily worn, and the thinner varieties are better than the heavier ones.

Sterilization may be accomplished by: (1) boiling for varying periods in soda solution or plain water, while wrapped in a towel. Some writers advise prolonged boiling for 15 minutes, but this soon destroys the gloves and makes them brittle. Brisk boiling for from one to two minutes is considered sufficient to destroy all germ life. They are then placed in sterile water or lysol solution (½ to 1 per cent.) which permits their being slipped on more easily. (2) By steam, after being powdered, wrapped up in a towel and placed in the autoclave. They can then be put on dry. (3) By chemical sterilization, for which the gloves are soaked in a 1:1000 bichlorid of mercury or other germicidal solution for an hour at least. The first method is the most practical and efficient.

As the gloves may be torn during the course of an operation or as it may be necessary for the surgeon to remove them, it is essential that the hands be disinfected as carefully as if they were not to be used. If a glove finger is accidentally punctured, a rubber finger cot may be drawn over the same.

Mickulicz introduced the cotton glove before that made of rubber was used. These have no advantages, and unless they can be kept dry, microörganisms can readily filter through the meshes.

Another ingenious substitute for rubber gloves has been recommended by Murphy of Chicago. He coats the hands with a solution of gutta percha in benzin or acetone which does not permit the secretions of the skin to escape, and moreover will not admit secretions, blood or pus into the pores or crevices of the skin. At the same time it does not interfere with the sense of touch or impair the pliability of the skin. These solutions in a strength of from 4 to 8 per cent. may also be used in place of the adhesive rubber dam formerly suggested by the same author. The operator's hands are first thoroughly washed with soap and water and then rinsed in alcohol. The rubber solution is then poured over both hands, care being taken to fill in all crevices around and beneath the nails. It may be quickly dried by exposure to the air, the fingers being widely separated. Although the coating is so very thin, it will resist washing with soap and water between operations, but if it wears off at the finger tips, a little fresh solution may be poured

over the defect. Removal is readily accomplished by washing with benzin and no deleterious effects observed to the surgeon's hands.

The preparation of the patient's skin is similar to that employed for the surgeon's hands, but varies somewhat with the character of the operation and the time of its performance. In accidents and emergencies, preparation of the skin to secure absolute asepsis must be sacrificed in many instances to other considerations bearing on the patient's condition. The methods generally needed for emergencies and accidents are described in works on "First Aid," and the following procedures are intended for deliberate operations where sufficient time is at hand. For minor surgical operations, scrubbing with soap and water is the most essential thing. This may be followed by washing with alcohol and ether, one or both. Painting the immediate area around the intended line of incision with tincture of iodin after the scrubbing, is also a most efficient aid in securing sterility of the deeper layers of the skin. Previous to laparotomy or other major operations it is usually well to begin the preparations the night before, when the region under consideration is thoroughly scrubbed with tincture of green soap and water, and then a soap poultice applied for six to eight hours. This is followed in the early morning by a compress wrung out of bichlorid solution (1:3000), which may remain in place until the time for operation. After the patient is on the table and usually after anesthesia is complete, the compress is removed and the skin again lightly scrubbed with soap and water, and washed with alcohol, ether and bichlorid solution. The soap poultice is made by soaking pads of sterile gauze in a mixture of green soap and water (3:1) which are then applied to the skin, covered by a nonabsorbent cotton dressing and held in place with a bandage. Where the epidermis is very thick (hands or feet) the poultice must be left on for a longer period of time than in other localities, in order to secure sufficient maceration of the layer. Where hair is present in the field of operation it must be carefully shaved off before the other preparations are begun.

The best known method for securing sterilization of the vagina is that suggested by Schimmelbusch. The cavity is scrubbed thoroughly with pads of gauze saturated with tincture of green soap and then thoroughly irrigated with a solution of bichlorid of mer-

cury, 1:2000. In place of the latter a solution of lysol (1:500 or 1000) or creolin (1:1000 to 500) may be used.

The bladder is rendered sterile with difficulty, and it is asserted by many that it is impossible to secure perfect asepsis of this organ, especially where a cystitis is present, even to a slight degree. The best known means at our disposal is by irrigation with a solution of potassium permanganate (1:1000) or boric acid (2 per cent.). Before introducing this solution the urethra should first be flushed out, and for several days or a week previous to the date of operation, the patient should receive some urinary antiseptic such as urotropin.

When operations on the rectum are contemplated, the patient should be given a purgative, followed by an enema several hours later. This is done on the previous day or morning before the operation, and is repeated an hour or so before the latter is begun. When the patient is on the table the region of the anus is again thoroughly washed and shaved, or the latter may have been done previously if no tenderness is present, and then the rectum is irrigated with boric acid solution. No bichlorid or other irritant solution should be employed. Contamination with feces may be avoided by pushing a pad of gauze, to which a string is tied, up into the rectum above the site of operation.

In order to sterilize the scalp it is necessary to shave off the hair before washing with soap and water. A green soap poultice should then be applied for a number of hours, and at the time of operation the usual soap and water scrubbing done, followed by washing with alcohol and ether.

The epidermis on the feet being very thick it should be thoroughly softened by soaking in hot water, after which a soap poultice is applied. The scrubbing must be vigorous in order to remove the macerated epidermis, but the remainder of the process is completed in the usual manner.

The preparation of the room for a major operation will only be considered in so far as it applies to doing this in a private house. If possible a well lighted room should be selected in one of the upper floors of the house and all unnecessary articles of furniture removed. It is best to take up the carpet and scrub the floor thoroughly, following this by the application of a strong bichlorid of mercury or carbolic acid solution. Where this cannot be done,

sheets moistened with a 1:500 bichlorid solution may be placed over the carpet in the neighborhood of the operating table. An ordinary kitchen table, thoroughly scrubbed, answers the purposes very nicely. This is covered with a thick blanket over which is placed a rubber sheet and over all a clean white sheet. Whatever articles of furniture remain in the room should be covered with sheets. One or two wash boilers full of thoroughly boiled water should be provided for making solutions, and other purposes. Plenty of towels and a number of sheets should be sterilized in one of the ordinary steam sterilizers, or if this is impossible they may be boiled in a wash boiler for half an hour and then allowed to cool. Basins intended for solutions and instruments may likewise be boiled or soaked in 1:500 bichlorid of mercury solution for several hours. It is well to remember, however, that it is not so much the preparation of the room, as it is the care with which the aseptic details of the operation itself are carried out, upon which depends the outcome at least in so far as healing without suppuration is concerned. Clean hands, clean instruments, clean ligatures and dressing materials, and a clean field of operation, with the patient in good physical condition and a minimum of shock are the essentials to good operative results.

The sterilization of ligatures and suture material is a topic which still remains to be discussed. These adjuncts to surgical technic are of two kinds,—absorbable and nonabsorbable. The former may be permitted to remain in place and be absorbed by the solvent action of the tissues; the latter require removal at some time subsequent to operation, for if permitted to remain, they act as a foreign body. The absorbable materials vary with regard to the time which is needed for their complete disappearance,—plain catgut being absorbed more quickly than chromic gut or kangaroo tendon. Catgut is now the most widely accepted suture and ligature material. It is prepared from the small intestine of the sheep and must be carefully sterilized before use as it contains numerous species of microörganisms which are very tenacious of life. Being pliable and yet fairly strong, it forms the ideal material for the purpose. Delay in absorption may be secured by impregnating the gut with chromic acid, so that this process may be extended from ten to thirty days. Among the numerous

methods proposed for sterilizing catgut, attention is briefly called to the following: In v. Bergmann's method the gut is loosely wound on glass spools and then soaked in ether for twenty-four hours. The ether is poured off and the following solution added: Bichlorid of mercury 10; absolute alcohol, 800; and distilled water, 200 parts. At the end of twenty-four hours the solution is changed, absolute alcohol being substituted for the bichlorid solution in which the gut may be permanently kept. If it is desired to have the gut soft, 20 per cent. of glycerin may be added. If any doubt exists as to the absolute sterility, the gut may be boiled for an hour in absolute alcohol, using a special vessel for the purpose to avoid the lighting of the alcohol. The most convenient means for storing the spools of gut is in wide-mouthed jars tightly stoppered.

Fowler's method consists in placing a number of strands of catgut in test tubes filled to one-half inch of the top with 95 per cent. alcohol. A wad of cotton is then introduced and the mouth of the test tube closed with a cork. These tubes are then inverted in a fruit preserving jar also filled with 95 per cent. alcohol, the top screwed on and the whole thing placed in a water bath and boiled for an hour.

For preparing chromic gut, the catgut after being sterilized is soaked in 5 per cent. carbolic solution, containing 30 grains of bichromate of potash to the quart, for 48 hours or longer depending on the size of the gut and the length of time which it is desired to delay absorption. It may be stored dry or in absolute alcohol.

Iodin catgut as suggested by Claudius is very easily prepared. It is very tough and thus far has been used with good success. It is made by soaking the gut loosely rolled on spools in a solution containing potassium iodid and pure iodine, each 1.0 gm.; and distilled water, 100 gms. Here it is allowed to remain for a week and may then be used immediately or stored in sterile containers. It retains its antiseptic properties after being inserted in the tissues, and may thus overcome any infection resulting from handling. In the skin it causes a slight reaction, and its absorption is also delayed, so that it cannot be recommended for this purpose.

Cumol gut, so called, has lately been used a great deal, and can be prepared by merely boiling the gut in this substance, in which it may also be stored.

Gynecology

POST-PARTUM HÆMORRHAGE AND ITS TREATMENT

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Strictly speaking, post-partum hæmorrhage means hœmorrhage after the labor is completely finished,—that is, after the placenta is delivered. Some authors divide the discussion of the various hæmorrhages occurring before and after the placental delivery, and also consider separately those hæmorrhages occurring later in the puerperium. Clinically it is feasible to discuss separately the late puerperal hæmorrhages, but it is impossible to divide those occurring in and directly after the third stage without destroying a rounded description of the whole subject. Few authors have forced the classification so sharply. The writer will consider in this paper only those hæmorrhages occurring during the third stage, and in the few hours immediately following the delivery of the placenta. While the term is not strictly correct, these hæmorrhages will be designated "post-partum hæmorrhages."

The reproductive function is the only one normally attended by hæmorrhage, and nature, by wise provision, fortifies the woman against the inevitable loss, and even over-provides so that she may survive a drain that would prove fatal in a non-pregnant female or a male. During pregnancy the total quantity of blood is increased, the fibrin is also greater in amount, these changes beginning after the fifth month. There is a slight leucocytosis which becomes marked (16,000) during labor. In healthy gravidæ these are the changes.

while in a sickly or anæmic woman pregnancy may induce a condition of chloro-anæmia. This point, first demonstrated by the hæmatologists has a strong clinical bearing.

During the separation and expulsion of the placenta, hæmorrhage is prevented by the retraction and contraction of the uterine muscle, and by thrombosis of the uterine sinuses. The more important factor in securing hæmostasis is the uterine action, since thrombosis may be absent and no hæmorrhage occur; and, conversely, thrombosis alone will not check a hæmorrhage unaided by the contraction and retraction of the uterus. In cases of sepsis the thrombi are dissolved and only rarely does a hæmorrhage result.

The sinuses in the uterine wall are simply blood spaces lined with a single layer of endothelium lying between the muscular lamellæ and muscle bundles. As the uterus empties, the muscular lamellæ become superimposed on each other, the muscle bundles crowd closer together. As a result, the blood sinuses are bent and twisted on themselves, effectually choking off the current of blood that flowed through them. There is even an effect on the larger vessels at the sides of the uterus after they enter its wall. The retraction of the uterine muscle does the actual work in this operation. The regularly recurring uterine contractions aid and support the permanent retraction.

Anomalies in the retraction and contraction of the uterus, and in the formation of thrombi in the uterine vessels will lead to post-partum hæmorrhage. Indeed, most of the hæmorrhages can be referred to these causes. Clinically we find another very potent factor—lacerations of the parturient canal.

The *causes* of post-partum hæmorrhage may be classified under three heads: (*a*) Lacerations of the parturient canal. (*b*) Atony of the uterus, or anomalous retraction. (*c*) Diseases of the blood or blood vessels.

(*a*) *Lacerations* of the genital tract play a much more important rôle in the production of hæmorrhage post-partum than is generally believed. Many hæmorrhages called atonic are due to tears in the genitalia. The writer was called to a case where the physician had used all known measures to stimulate the uterus to contract, when the woman was pulseless from a bleeding clitoris tear. One suture stopped the flow.

Laceration of the clitoris and of the bulbi may give rise to even fatal hæmorrhage, as may also a ruptured varix, though both are rare. Perineal tears seldom bleed enough to require immediate suture, and vaginal tears also are rarely attended by much hæmorrhage. They may, however, require suture or packing, and when they do, packing is the best method because the field is so inundated with blood that it is impossible to see where to place the suture. High vaginal tears, extending into the fornices, may lay open the bases of the broad ligaments and may bleed furiously. They are usually combined with cervix tears, or are the extension of cervix tears.

A laceration of the cervix extending into the broad ligament may not bleed if the tearing is gradual and not forced. Such findings are occasionally made after spontaneous labors or dilatation with one of the crushing dilators (the Bossi, de Seigneux, Harris etc.) which are now quite popular. Usually, however, a deep cervical tear gives rise to serious hæmorrhage. Deep cervical tears are most likely to occur if a rapid delivery is made before the cervix is completely effaced or shortened, that is, drawn up into the body of the uterus. Even after the cervix is completely dilated a laceration of the upper fibres may result if the extraction of the child is forced during a spasmodic contraction of the lower uterus and upper cervix. Sometimes the head passes through the cervix and the contraction ring closes down around the neck of the child. Forcing the delivery in such a case may cause the shoulders to tear the edge of the contraction ring. In the last three varieties of cervical laceration the tear is more likely to be a longitudinal split of the cervical canal, usually lateral, extending more or less deeply into the broad ligament, and indicating that if it went much further it would have to be called a rupture of the uterus, incomplete or complete, depending on whether the peritoneal cavity were opened or not. Without doubt many cases of so-called post-partum hæmorrhage are in reality ruptures of the uterus, as likewise are many cases of "shock" after labor. A laceration of the uterus, occurring during an operation or spontaneously, gives rise to internal and external hæmorrhage, and such cases are not to be considered in this paper.

When the cervix tears after complete effacement but before complete dilatation, few large blood vessels are torn, unless the tear

extends into the bases of the broad ligament. Such cases are relatively easy to treat (Fig. 1).

Special emphasis must be placed on the lacerations of the cervix and lower uterine segment occurring in placenta prævia. It is usually thought that the frequency of post-partum hæmorrhage in placenta prævia cases is due to uterine atony. As a matter of fact atony is rare in placenta prævia; indeed there is often too strong uterine action. The branches of the uterine artery supplying the lower uterine segment are given off from the uterine above the contraction ring, and, therefore, are somewhat compressed by the contracting uterus before entering the relaxed lower uterine segment. While one would expect a placental site, located out of the active contracting zone, to permit free oozing, this is, to a large extent, prevented by the peculiar situation of the blood supply. Hæmorrhage is therefore due to some other cause, and this will usually be found to be a laceration. The extreme vascularization of the lower uterine segment and cervix, due to the placental implantation, permits even a tiny tear to bleed profusely. Since the veins and sinuses are situated close to the surface, a laceration one eighth of an inch deep may cause a fatal hæmorrhage. Indeed, hæmorrhage from a cervical tear in placenta prævia is a most appalling complication. It was carefully considered by the writer in an article on "Placenta Prævia" in the January INTERNATIONAL CLINICS.

(b) *Insufficient retraction or contraction* of the uterus or anomalies of the same, cause most of the fatal post-partum hæmorrhages. They may be associated with any of the lacerations previously described, and when the tear is situated in the lower uterine segment the absence of a firm restraining action of the uterus is apparent.

It is not feasible to try to separate anomalies of retraction from those of contraction. Normally the two functions coöperate, and in practice we find that where the uterus does not contract well it does not retract well. We will call a uterus atonic when the contraction and retraction of its muscle is insufficient or irregular, though etymologically and strictly, other terms should be used.

The causes of atonic hæmorrhages are general and local. Generally weak women, those of excessive cultivation, who often have

FIG. 1.

Diagram of parturient canal showing the common sites of lacerations. They may be single or multiple, small or extensive.

poorly-developed genitalia, and generally poor innervation, have slow labors and a greater tendency to atony in the third stage. In some families a tendency to post-partum hæmorrhage is transmitted. This is an entirely different condition from hæmophilia, which is to be considered later. Some women have atony of the uterus in successive labors. This may be due to a persistence of a local cause. Blondes and red-haired women, in the writer's experience, are likely to have hæmorrhage post-partum, but how much is dependent on the constitution of the blood, it is impossible to say. Myocarditis, valvular disease, and pulmonary affections are said to favor atonia uteri, but the writer has had little trouble with it in such cases. Myocarditis seems most likely to predispose to atonia uteri. Chronic nephritis has been given as a cause of atony, a degeneration of the uterine muscle being directly active.

The use of chloroform during labor undoubtedly increases the tendency to hæmorrhage, though this action is not constant.

Of local causes there are many: Over-distention of the uterus, by twins, polyhydramnion; a large or diseased fœtus, or an accumulation of blood (abruptio placentæ) (the elongated muscle fibres need time to accommodate themselves to the rapidly diminishing uterine cavity so that rapid emptying of such a distended uterus nearly always causes hæmorrhage.) Abnormal shape of the uterus, bihorned uteri, uterus arcuatus, septus, etc.; distortions of the uterus by adhesions (rarely); distortions by tumors, as fibroids (commonly); old scars in the uterus, after Cæsarean section, or ruptures,—all these interfere with the contraction and retraction of the organ and allow the sinuses to remain open, and from these more or less profuse hæmorrhages occur.

Metritis is given by J. Veit as a cause of atony. That endometritis may cause post-partum hæmorrhage is true beyond doubt. The atony in such cases is secondary to the retention of bits of placenta or membrane or blood, due to the endometritis. The retention of tiny bits of placenta in the uterus is not rare. Usually they come away in the lochia, rendering the discharge more profuse and fœtid. Occasionally these bits form a mass with thickened decidua and blood-clot, in which case they give rise to atonic hæmorrhage. The adhesion of a piece of placenta to the uterus always gives

trouble, sooner or later. It prevents the complete retraction of the uterine wall immediately adjoining, and thus permits blood to escape from the open sinuses. The blood accumulates in the uterus, distending it, and thus a general relaxation occurs. The cause of retention of a piece, or of the whole of the placenta, is endometritis, (Simpson, Martin, Cohnstein, Meyer-Rüegg et al.). In these cases microscopic examination show the chorionic villi in direct contact with the muscle fibres. The serotina have completely disappeared. Hæmorrhages early in pregnancy may cause pathological adhesion of the placenta. A previous curettage which is too deep may also cause the same thing. It may be impossible to remove the adherent placenta, the villi having established such an intimate connection with the uterus, and these cases are often fatal, since tamponade may not stop the hæmorrhage, and extirpation of the uterus, the only certain resource, may not be done soon enough or quickly enough to save the exsanguinated woman.

Retained membranes seldom cause atony or hæmorrhage. They may, however, if a piece becomes detached and encloses a blood-clot. This forms a foreign body, and the uterus may relax over it. The thickened decidua near the edge of the placenta, in cases of endometritis, is likely to bind the chorion to the mucosa, and it may act like a piece of retained placenta. Not seldom a tiny placentula succenturiata or a placenta spuria is torn off with the membrane. In such cases the delivered placenta, on cursory examination, appears complete. The missing membrane may lead to the discovery of the absent piece of placenta. The accumulation of blood itself in the uterus may keep up a uterine atony. If the uterus is firmly squeezed, thus evacuating the clots, the bleeding ceases definitely. If all the clots are not removed the uterus will soften and balloon out again. One may err with too much zeal in such a case, and by repeatedly squeezing out clots, exsanguinate the woman. The uterus cannot fully retract on itself so as to obliterate its cavity, and to fill the vacuum, blood flows in. The treatment of such a case is the single evacuation of the clots, followed, if such treatment is not sufficient, by uterine tamponade.

Multiparæ suffer more from atony than primiparæ, this being especially true if the births followed closely one on the other. The uterus has grown tired. Women who have suffered from subinvolu-

Fig. 2.

Contraction at the ring of Bandl,—so-called hour-glass contraction. Note the hæmorrhage behind the loosened placenta.

Fig. 3.

Partial adhesion of the placenta. Anomalous contraction of the uterus.

tion are more exposed to post-partum hæmorrhage. Women who go too long after the natural termination of pregnancy—cases of delayed labor—are likely to have an atonic third stage. The fact that they go over time shows that the uterus is indolent, which condition may persist throughout labor. Then, too, the overdistention of the uterus from the large child may operate. In cases of "missed labor" such uterine atony is most marked. It is due to structural changes in the musculature.

Atony of the placental site is a rare cause of post-partum hæmorrhage. The rest of the uterus contracts well, but the placental site is flaccid and bleeds freely. The hand inside the uterus feels the relaxed wall, the outside hand notes a depression in the uterus. Marked cases of this condition are rare, but milder cases are not so very uncommon.

A uterus that is tired from prolonged labor may default in the third stage. If the pains have been strong during the first two stages they are likely to continue strong in the third. If weak, nagging, and ineffectual, then atony may be expected in the delivery of the placenta. It is then not so much hard labor as protracted labor that invites post-partum hæmorrhage. In these protracted labors the cause is rarely to be found in irregular uterine contraction. Rings of contraction form and parts of the child are locked in a position so that it can make no forward progress. These same contraction rings are formed after the delivery of the child and imprison the placenta. This is the "hour-glass contraction" of older writers. (Fig. 2.) It occasionally is the cause of hæmorrhage during the third stage.

Partial adhesion (Fig. 3) of the placenta may cause hæmorrhage. Irregular uterine action is responsible for anomalies in placental separation, and the partly separated organ is incarcerated by the uterus, giving rise to very dangerous hæmorrhage. A full bladder is a not infrequent cause of retention of the placenta and of post-partum hæmorrhage. The distended bladder compresses the cervix, allowing blood to accumulate, and, owing to the connection between the two organs, the uterus cannot contract so well. Many cases of hæmorrhage post-partum cease immediately when the bladder is emptied, and often manual removal of the placenta is rendered unnecessary by simple catheterization.

A not-to-be-neglected cause of anomalous uterine action is improper conduct of the third stage. Too much massage or misdirected rubbing in normal cases, pulling on the cord, premature attempts to remove the afterbirth, by Credé expression, the administration of ergot during labor and before the placental delivery, all disturb the normal course of the third stage, produce anomalous uterine action, break the retro-placental blood clot, favor partial adhesion of the placenta, and incarcerate the organ, causing hæmorrhage. While it is not true that all post-partum hæmorrhages are preventable, it is true that the majority of hæmorrhages are caused by improper conduct of the third stage of labor. The third stage of labor is more important than the other two. Obstetrical textbooks neglect the consideration of this important topic. It requires more knowledge and skill to conduct the third stage intelligently and properly than either the second or the first stage. Over haste in the delivery of the placenta, before the uterus has had time to retract fully on itself, to get, as it were, a good grasp on itself, is responsible for many severe floodings and even deaths.

Finally may be mentioned precipitate labors and rapid artificial delivery. Post-partum hæmorrhage is uncommon after precipitate labors, but it does occur and it may even be fatal. In the latter cases the question arises, is not the hæmorrhage due to laceration of the uterus? Uterine atony may occur.

Rapid artificial delivery is frequently followed by uterine atony. Indeed, often the indolent uterus necessitates interference, and naturally the condition persists into the third stage. In normal cases, dragging the body of the child out forcibly after the head is born is a frequent error of technique, often leading to evil consequences for the mother and for the child. The same is true of rapid forceps deliveries and extraction by the breech. The uterus *must* have time to accommodate its muscle walls to their diminishing contents. The occurrence of lacerations with these rapid deliveries must not be forgotten.

(c) *Abnormal blood states,* or diseases of the blood vessels, may cause severe and even fatal post-partum hæmorrhage. Hæmophilia is rare, and while it usually causes severe hæmorrhage, death seldom results. Ahlfeld (*Zeitschr. für Geb. u. Gyn.,* Bd. xlvii, S. 224), reports a fatal case where biochemical analysis of the blood proved the

diagnosis. The writer has had 4 cases of hæmophilia in labor, 1 of which died. The fatal case was one of abruptio placentæ with continuance of the hæmorrhage post-partum in spite of tamponade, etc. There were numerous hæmorrhages into the tissues, at the sites of hypodermic needle punctures. Fellner ("Innere Krankheiten," Wien, 1903, S. 180) reports 6 cases of severe hæmorrhage in hæmophiliacs. Is there a temporary hæmophilic condition of the blood? The writer believes that there is, and it may be due to syphilis, malaria, leukæmia, certain metallic and alkaloidal poisons, etc. (See Eichhorst, "Lehrb. der Path. u. Ther.")

Scurvy, morbus maculosis Werlhofii, certain forms of anæmia, may all lead to profuse hæmorrhage. Basedow's disease may lead to intra- and post-partum hæmorrhages. Blondes, especially those of pasty, watery constitutions, have thin blood, and are more frequently attacked by post-partum hæmorrhage. There is much that is hazy and uncertain in the discussion of this group of causes, but the writer is convinced that a study of the blood states in pregnancy and of the pathology of the blood vessels and blood-making organs, will show many interesting facts and explain many conditions beside the single one under consideration.

SYMPTOMS

The symptoms of post-partum hæmorrhage are general and local. The general symptoms are those of hæmorrhage, and depend on the amount of the blood lost, the rapidity with which it flows away, and the physical condition of the patient beforehand. There is also the element of shock involved. Women vary very much in their ability to stand hæmorrhage. Chloro-anæmics, leukæmics, women suffering from malarial or certain forms of cachexia and typhoid, are acutely endangered by even a moderate loss of blood. Women who have lost blood during pregnancy are in poor condition for a loss during labor, the blood-making organs being exhausted. Marked examples of such individuals are seen in cases of placenta prævia. A sudden severe loss is badly borne because of the attending shock. When the shock is overcome the patient rapidly regains her color. It is often difficult to distinguish between shock and hæmorrhage, or how much of the symptoms to ascribe to each condition. Sometimes the blood gushes out in a stream that appals the accoucheur, and may kill the

woman in a few minutes. These are cases of atonia uteri usually, and are fortunately rare. The blood may come out in a continuous stream, or interruptedly, mixed with clots. A large accumulation of blood may take place behind the placenta, balloon out the uterus, and when the hand is laid on the abdomen the whole mass is expelled. This should not frighten the accoucheur, because this blood is already lost to the woman. Again the hæmorrhage may be irregular, the uterus filling up and emptying itself several times. Occasionally a slight hæmorrhage at the start, which one considers harmless and easily stopped, continues and interferes with the best work of the accoucheur; then, again, there is one gush, after which no more blood escapes. Continuous hæmorrhages usually come from lacerations. Hæmorrhage may be internal or external, or both. If the os is occluded by placenta, membranes or clot, the blood distends the uterus, and symptoms of anæmia develop without visible hæmorrhage. Usually, however, the bed is flooded with blood, and this gives the first warning.

The symptoms of hæmorrhage in the usual order of their appearance are: faintness, dizziness, air hunger, yawning, nausea, restlessness, thirst, præcordial anxiety, general fear of impending dissolution, vomiting, fainting collapse, convulsive twitchings, and, as death nears, involuntary bowel movements, and coma. These symptoms are noticed in their order if the loss of blood continues. In an anæmic person they come on sooner and more pronouncedly. The physician observes in the patient these signs as the hæmorrhage continues: Rapid pulse, pallor of lips and skin, coldness of tip of nose and face and extremities, cold sweat on brow, sunken eyes, rapid respiration, and all the symptoms just noted. The pulse is rapid and weak, of small volume, but it may be slow and weak. Sometimes the pulse holds up well, even when there has been much blood lost, until a certain mark being passed, the grasp of the vessels on the diminishing blood is lost and the heart gives way. These cases should be recognized by the accoucheur, so that he may not presume too much on the capacity of the woman, resting his judgment on the pulse. The pulse becomes more rapid and weaker, finally is irregular and disappears at the wrist. It is our best indicator of the severity of the bleeding.

It is astonishing how much blood a woman can lose and survive.

Ahlfeld (*Zeitschr. für Geb. u. Gyn.*, Bd. li, Heft 2) confined several thousand women on a bed with a hole through the centre, underneath which was placed a glass graduated pail. He found that in a normal labor the loss of blood should average 435 grams, that pathological labors showed an average of 677 grams, the average blood loss of all labors being 505 grams. Ahlfeld therefore concludes that a loss up to 800 grams is normal, and that a healthy woman of average weight may safely lose a liter, which is a little more than a quart. The writer has his own opinion about this, and will express it under Prognosis. Ahlfeld found the first symptoms followed a loss of 1 liter, pallor and rapid pulse; as the flow continued up to 1,600 or 1,800 grams, the surface of the patient grew cold, sweat appeared, and thirst, dyspnœa, yawning, dizziness, and faintness were to be observed. Occasionally death occurred, with a loss of less than 2 liters. In 6,000 labors there were 14 hæmorrhages over 2,500 grams (5 pints), 1 of which was over 3,000 grams. All the patients recovered. A loss of 3,800 to 4,000 grams (a gallon is considered absolutely fatal. No doubt the danger of severe blood loss post-partum appears greater than it really is, but since one cannot, beforehand, foretell the woman's ability to stand hæmorrhage, is is prudent to take alarm at small hæmorrhages and quickly institute means to check them.

The local findings vary with the cause of the hæmorrhage. In cases of laceration these will, of course, appear prominent to the careful examiner. The hæmorrhage is continuous from the start, and the flow usually begins as soon as the child is born. Blood may not show for some time outside, even from a laceration. With a laceration there is nothing of an unusual nature in the separation and expulsion of the placenta, unless the hæmorrhage demands interference.

In cases of atony of the uterus the abdomen feels boggy all over, and one misses the hard globular uterus. When the hand is borne heavily on the belly, blood gushes from the genitals. In cases of partial atony with constriction rings locking in the placenta, the uterus is smaller, but filled partly with blood under some elastic tension. If the placenta is out and a bit of clot or other mass in the uterus prevents full retraction and contraction, the uterus is small but releases and contracts irregularly, each change being attended with a flow from the genitals.

The first blood lost is thick and deep red, later it grows paler and more watery. It sometimes loses its clotting power when it has become thin. The writer believes the clotting power of the blood varies in women and at different periods of life. Sometimes the blood is dark, like port wine, and the clots are black, or it has an indication of being "lake-colored"—that is, translucent. These cases are pathological, and one must be on his guard against them.

THE DIAGNOSIS

That a parturient is bleeding should not be brought to the accoucheur's notice by the symptoms of hæmorrhage. Ideal obstetrics demands that no woman should lose sufficient blood to give rise to symptoms. The diagnosis then has to do almost entirely with determining the cause of the bleeding, with a view of procuring the quickest hæmostasis. Once in a while differential diagnosis will be required between hæmorrhage and shock. The differential diagnosis of post-partum hæmorrhage from rupture of the uterus, rupture of any of the abdominal viscera, pulmonary embolism, etc., occasionally has to be made, but the limits of this paper forbid such consideration.

It is the writer's practice, in the presence of a post-partum hæmorrhage, not to waste valuable minutes in completing an accurate diagnosis of the site of the bleeding. If a forceps operation has been performed, one will suspect a laceration of the vulva or vagina or cervix; if version and breech extraction, laceration of the cervix or uterus. One may discover a laceration as soon as it is made during the latter form of operative deliveries. Before the placenta has been delivered it is impossible to determine whether the hæmorrhage comes from anomalous separation of the organ or from a laceration in the parturient canal.

A firm massage is administered to the uterus, and, unless a laceration is the cause, the hæmorrhage ceases when the uterus contracts. At the same time the clitoris, vulva and introitus are rapidly inspected for tears. A bleeding point is easily found if it is outside, if inside one needs specula; but it is not wise to wait for them. It is best to arrange immediately for definitive measures. The placenta is removed by Credé expression. If the uterus contracts and retracts, the hæmorrhage now ceasing, the cause lies in a

pathological third stage mechanism. If the uterus remains firm and hæmorrhage keeps up, the probability is that a laceration is the cause. If the uterus releases and balloons out under the hand, atony is the cause, and there is some reason for the atony. Many points in the diagnosis were mentioned under Symptoms, so that repetition is unnecessary.

As soon as the placenta is delivered, before it is removed from the bed, it must be carefully inspected to determine if any portion is missing. This is done while the hands are still sterile, as it may be necessary to enter the uterus.

If the placenta cannot be expressed by the Credé method, it is either adherent wholly, or in part, or incarcerated. If the placenta is wholly adherent there is no hæmorrhage, unless a brusque Credé expression loosens one portion. If the placenta is partly adherent, the uterus usually contracts irregularly, which may be determined by the outside hand, and the hæmorrhage is continuous. With an incarcerated placenta the uterus is usually hard, hæmorrhage is moderate, and on internal examination one feels the strictured internal os. A partly adherent placenta may be locked in the uterus by a constricted internal os. In an atonic uterus the internal os is not easily defined, and the uterus in general presents no firm body. When the uterus is large, boggy, hardly definable, sometimes reaching the liver, one knows that it is full of blood, or blood and placenta. A brisk abdominal massage causes the uterus to contract and to expel clots and fluid blood. When atony of the uterus complicates a laceration, it may be impossible to determine the extent of the tear till the uterus has been tamponed. Thus may be seen how the therapy and diagnosis go together.

THE PROGNOSIS

Few women die from post-partum hæmorrhage. Statistics, proverbially unreliable, show 1 death in 1,000, 2,000 or 3,000 labors. The Chicago Lying-in Hospital Dispensary service has had 8,100 consecutive cases without a death from post-partum hæmorrhage. There have been quite a few serious cases of bleeding, and 3 women were saved from apparent death. The worst hæmorrhages come from atonia uteri, combined with laceration of the cervix. Placenta prævia deserves mention in this connection.

Atonia uteri has not been so commonly noted as cervical tear, but either may cause alarming losses. Vaginal and vulvar tears usually are not serious, but in one case of clitoris tear the patient nearly died, the attendant treating her for atony of the uterus.

Retained placenta, or parts of it, cause alarming hæmorrhages, which are usually completely controlled on removal of the cause. If the entire placenta or part of the placenta is densely adherent to the uterus the case becomes formidable, for it may be impossible to get the organ off the uterine wall, and unless properly treated the patient dies of hæmorrhage, or in the attempts to remove the offending piece of placenta the finger bores through the uterine wall. Hæmophilic tendencies increase the danger of hæmorrhage, and may cause death. Few hæmophilics die, otherwise the race would become extinct, for the tendency is transmitted through the mothers.

It is difficult to foretell how a bleeding once begun will end. Sometimes a moderate oozing baffles all treatment, and lays heavy burdens on the anxious accoucheur. Again the bleeding may start in furiously and stop suddenly. In the slow, insidious hæmorrhages, a piece of placenta in the uterus, or hæmophilic tendencies may be the cause, and the oozing will not cease till proper treatment is instituted. It is advisable to stop all hæmorrhages early, because, the thinner the blood the harder it is to cause clotting: further, there are late effects of hæmorrhage, soon to be mentioned. There are, too, certain unknown factors operating in such cases; some women can lose enormous quantities of blood and still recover, others die from smaller losses. The warning to be drawn from this is apparent.

There are remote dangers in these cases. Thrombosis and embolism are commoner after severe hæmorrhage. Sepsis is commoner, first because the exsanguinated woman has insufficient resisting power against infection, and secondly because the accoucheur, in his haste and anxiety at the time of the hæmorrhage, neglects his antiseptic precautions. This is a real danger.

Hæmorrhage into the retina and blindness have been noted as a sequel, as well as chronic diarrhœa and, perhaps, gastric ulcer. Permanent debilitation of the blood is not an uncommon after-effect, and various blood dyscrasiæ, as leukæmia, have been said to be thus caused. Permanent neurasthenia has resulted in the writer's observation, and in minor cases, more or less prolonged invalidism.

Even in cases of so-called normal losses, 800 grams, the patients do not make as satisfactory recoveries as those who lose less than 400 grams. The women are longer abed and are very slow to regain their strength. Lactation is interfered with, Ahlfeld to the contrary notwithstanding.

In tubercular women, and those from such families, the prognosis is serious from the viewpoint of late effects, and redoubled precautions against hæmorrhage ought to be instituted.

In leukemics or other blood-sick women the normal loss at labor may be directly fatal.

Thus it is apparent, that while nature has thrown much protection around the parturient woman, it well behooves the accoucheur to afford her all the additional advantages his art possesses.

TREATMENT

Prevention plays a most important rôle. It begins during pregnancy, and an accurate knowledge of the woman's constitution is necessary. One must know if an endometritis has been present before pregnancy occurred, since a diseased endometrium often causes adherence of the placenta and membranes, or even anomalies in the muscle. Hæmorrhagic tendencies must also be inquired after and a family history of post-partum hæmorrhage may be obtained. One woman reported by Michäelis had profuse hæmorrhages in every labor, " blood coming out as from a barrel," so that at every delivery it was necessary to remove the placenta at once on the delivery of the child. The writer has a patient who had post-partum hæmorrhage in two labors and whose mother and sister are similarly affected. In the latter class of cases the writer administers iron, arsenic, and strychnia in minute doses during the last month of pregnancy, and insists on a great deal of out-door living.

Where there is reason to suspect hæmophilic tendencies gelatine pudding is ordered as a daily refreshment in the last month and calcium chloride in 20-grain doses every three hours is administered as soon as labor begins. Where there is reason to suspect endometritis, or there is a history of previous floodings, extra care is given the third stage, and preparations are made to remove the placenta immediately and stop the hæmorrhage definitively without waiting for a severe flow to force the necessity of interference.

During labor the patient must be supported with food; she should not become exhausted by prolonged travail, and operative help must be given her before the uterus has become tired out. On the other hand, one must obtain complete cervical dilatation, to avoid hæmorrhage from its laceration, and one may not extract too early before the uterus has been in regular strong action, or too rapidly, for then the uterus would not retract quickly enough to prevent hæmorrhage.

In normal deliveries after the head is born the uterus should be allowed to expel the trunk, which must not be dragged out rapidly. Have a hand follow down the fundus uteri as the fœtus escapes, massaging only when it is necessary. With twins, hydramnion and large fœtuses, slow delivery is necessary. Be sparing in the use of chloroform.

The treatment of the normal third stage should be properly carried out. Too much massage of the uterus, too early attempts at Credé expression, traction on the cord, all produce irregularities in the separation and expulsion of the placenta, causing hæmorrhage, and often the necessity for manual removal and as a result occasional infection. Credé and the Dublin school originally recommended the removal of the placenta by expression immediately after the delivery of the child. Ahlfeld instituted a purely expectant treatment of the third stage, the expulsion of the placenta being left entirely to nature. If after several hours of waiting the placenta did not come, "early expression" was resorted to. The writer some years ago experimented with various methods of treatment of the third stage, and concluded that both the above procedures were largely wrong and the best way would be to combine the good points of both.

The hand follows down the retracting uterus, exercising no massage, but only control. When the feet of the child escape the vulva the amount of blood and the condition of the uterus are noted. If there is no hæmorrhage and if the uterus is firm—no massage; contrary conditions—gentle massage. The thumb lies in front, the fingers behind the fundus, and circles are drawn rapidly on the uterine wall near the top, away from the cervix. As soon as the uterus contracts, the hand lies quietly watching the action of the organ.

If the uterus rises up and *balloons out with blood*,—massage.

In normal cases these accidents are absent, so that all that is required of the attendant is that he rest his hand lightly on the belly, noting the various phenomena of the third stage as they develop. When the attendant is sure that the placenta has separated and lies free in the lower uterine segment and upper vagina, "early expression" may be performed. This function requires from ten to forty minutes, or even several hours, but there is no benefit in waiting after the placenta has left the contracting portion of the uterus. The reason for setting an arbitrary limit of thirty or forty minutes for the expression of the placenta is that most men cannot determine when the placenta has been safely loosened, and to avoid trouble a reasonably safe time is allowed. In the writer's practice the average length of time for the separation and extrusion of the placenta is twenty-five to thirty minutes.

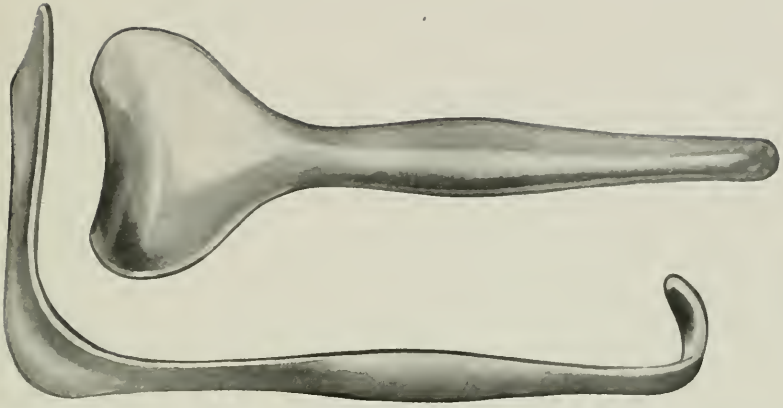
The signs which indicate that the placenta lies outside the fundus uteri and may safely be expressed are: (1) The uterus rises up toward the liver; (2) the uterus changes from a globular to a flattened pear-shape, the upper edge of the fundus growing sharper; (3) there appears above the pubis a soft boggy mass, the placenta behind the bladder; (4) the cord advances two to five inches from the vulva; (5) the vessels in the cord which became tense with each uterine contraction do so no longer. "Early expression" (Ahlfeld) differs from Credé expression. In the former the hand grasps the uterus and forces it down on the loosened placenta in a direction parallel with the long axis of the body, thus pushing the placenta out of the vagina. The uterus is used simply as an intermediary. In Credé expression the same motion is made, but in addition the thumb and opposed fingers are brought together, thus squeezing the placenta *out of the uterus,* like the "pit out of a cherry."

Feeling sure that the placenta has left the contracted uterus, then, early expression is performed; if it fails, a mistake has been made, the placenta is not yet separated; one must wait until this occurs. In twenty minutes another expression is attempted, and if this fails, Credé expression is tried. If again this fails one concludes that the placenta is adherent and, in the absence of hæmorrhage, either internal or external, one waits an hour longer. It is then justifiable to remove the placenta manually unless the expression performed now is successful.

Preparations for the treatment of hæmorrhage should be made in all labor cases. Half the battle is won if the accoucheur is prepared for all emergencies. A physician assuming the responsibilities of a case of labor should have at his fingers' ends all the methods of controlling hæmorrhage, both obstetrical and surgical, and he should carry with him to the case all things necessary for all such complications. The obstetrical kit should contain needles, short, round, full curved, of several sizes, needle holder, broad perineal retractors (Fig. 4), vulsellum forceps (Fig. 5), (these for repair of the cervix,) uterine packing forceps (Fig. 6), uterine douche-point (Fig. 7), several ordinary artery and tissue forceps. These instruments should be carried in a pan in which they may be quickly boiled when needed. Some accoucheurs carry such instruments already sterilized, or boil them at every labor just before the child is born. An apparatus for injecting salt solution hypodermically, at least a proper needle (Fig. 8), should never be wanting in the obstetrical satchel, and a jar of gauze one-half yard wide and twelve yards long should also be taken to each case. Facilities for giving a hot uterine douche should always be at hand. All during the labor the supply of hot sterile water should be kept up, and a hypodermic syringe in working order, with aseptic ergot, etc., should be at hand. Forearmed is forewarned.

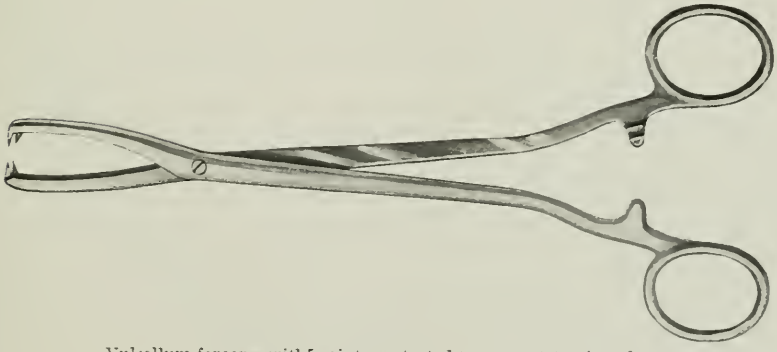
The gauze above mentioned is prepared as follows: sterile gauze as it comes from the surgical supply houses is cut into strips one-half yard wide and twelve yards long; the selvedge and raw edges are rolled inward and the gauze thus folded lengthwise into rolls as large as the finger; they are tied in separate bundles, washed in running water, and boiled thirty minutes in a ½ per cent. lysol solution; the bundles are then run through a sterilized clothes wringer, using sterilized gloves; each strip is then packed into a sterilized glass jar, using sterilized gloves, after which the jars are closed and steamed for two hours in the sterilizer, not under pressure; next day the jars are steamed again for two hours, and just before they have cooled off the covers are sealed with boiled paraffin and wrapped in sterile paper. Thus prepared, gauze will remain sterile and moist for years. If it is packed properly, in circles from the bottom of the jar, the tamponade is made directly from the container (Fig. 9). For years the author carried a bottle of sterile vinegar in the satchel,

FIG. 4.



Perineal retractor. Size of blade 3 inches long, 2¾ inches broad. Another one slightly smaller is also used.

FIG. 5.

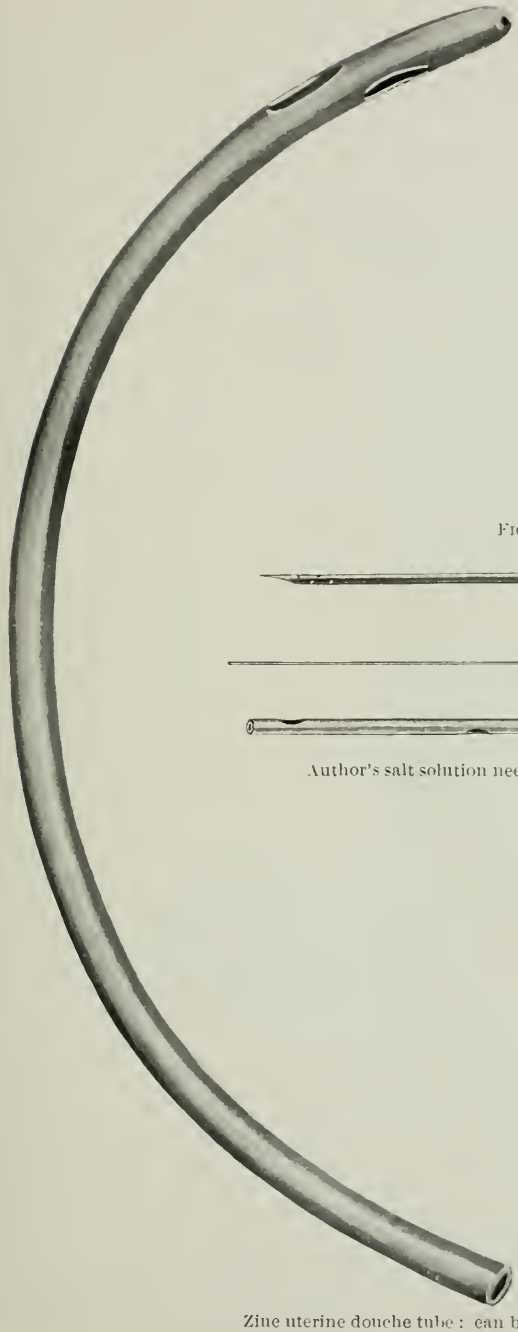


Vulsellum forceps, with points protected to prevent tearing gloves.

FIG. 6.

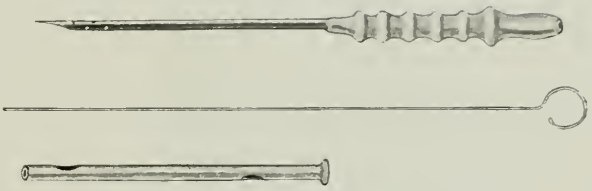
The long uterine packing forceps.

Fig. 7.



Zinc uterine douche tube: can be bent.

Fig. 8.



Author's salt solution needle, with point protector.

FIG. 9.

Uterine packing gauze as it is packed in glass jars.

and later a package of gelatine was put in its place, but never has occasion arisen to use either. On two occasions, in hospital practice, gelatine soaked tamponade was employed.

Treatment of Hæmorrhage during the Third Stage.—When a flooding follows the delivery of the child our first move is to grasp the uterus and massage it vigorously. As a rule, as soon as the uterus contracts the hæmorrhage ceases. At the same time the attendant inspects the genitals to find a laceration that may be bleeding. If the uterus contracts and the hæmorrhage continues, an internal examination should be made to see if there is a cervix tear. Should the bleeding continue it is necessary to remove the placenta whether a tear is found or not. Massage the womb vigorously, and in the moment of contraction perform Credé expression. This may stop the hæmorrhage; if not, it will be needful to remove the placenta manually. Before attempting the Credé, one must empty the bladder by catheter, bring the uterus to the median line and squeeze only when the organ is contracting. It is useless and very dangerous to squeeze a soft uterus.

Having decided that it is necessary to remove the placenta manually the patient is brought across the bed, with the buttocks well over its edge, the assistants, physician, nurse, husband or neighbors, hold the legs in the lithotomy position, and the accoucheur sees that his instruments, basins with antiseptic solutions, gauze for packing the uterus, and a hot douche are all prepared and at hand. If the accoucheur has his obstetrical technic well developed and his assistants and, most important of all, his clientèle properly trained, it is possible to be so prepared for labor cases that a change from the bed to the kitchen table may be made in a minute or two. Usually, however, the exigencies of the case will force the physician to operate with the patient across the bed, but, wherever it is possible, one should do these things on a table.

A careful disinfection of the external genitals and the lower vagina should now be made. No hæmorrhage is so dangerous that such preparation may be neglected. The danger of sepsis would thus replace that of hæmorrhage. The parts have already been cleansed, and they have been well washed with antiseptic solutions during the delivery. Before inserting the hand the externals should be again liberally sponged with 1 per cent. lysol solution, and then

with 1–1,500 bichloride. The vagina should be well flushed out with 1 per cent. lysol solution, using large cotton or gauze sponges. The use of rubber gloves cannot be too warmly recommended for obstetrical practice, and especially for this dangerous operation of invading the uterine cavity they are almost indispensable. The gloves should be long, should fit well, and they should be drawn on, dry, over sterile hands. A sterile towel is laid on the abdomen and the hand put into the vagina, avoiding the anus and perineum. The left hand is usually found to be most adapted for internal work. The fingers spread out in the cervix so that the membranes and cord fall into their grasp, then the tips of the fingers are brought together to form a cone for passage of the contraction ring (Fig. 10). It is seldom necessary to use force in passing this point. If ergot has been administered or if the contraction ring has closed down, it may be necessary to press the fingers firmly through the constriction. The inside hand seeks the edge of the placenta, outside the membranes, and the hand on the belly forces the uterus down over the inside fingers (Fig. 11). With a gentle combined movement the placenta is separated from its bed. It is usually very easy to separate the organ; but if it is pathologically adherent, tough strands will have to be sawn through by the fingers supported by the outside hand. A warning not to bore into or through the uterus must be given because this is sometimes surprisingly easy. After the placenta has been cleanly separated the two hands give the uterus a brisk massage, and a contraction will expel the hand containing the placenta. It is better to let the uterus expel the hand with the placenta than to drag the latter out. After washing the blood off the hand and again sponging the parts with antiseptic solution, the hand is once more inserted and the whole interior of the uterus carefully inspected. One must be absolutely certain that the uterus is perfectly empty. Care should be taken to remove all the membranes and thickened decidua. The rubber gloves are so slippery that shreds of decidua cannot be grasped. One misses the sharp finger nail in such cases. The writer uses a piece of gauze for the purpose of cleaning off the decidua and membranes as in Fig. 12. One may now give a hot 1 per cent. lysol intra-uterine douche, which usually causes firm contraction and retraction. A hypodermic of ergone, ergotale, or aseptic ergot is also administered.

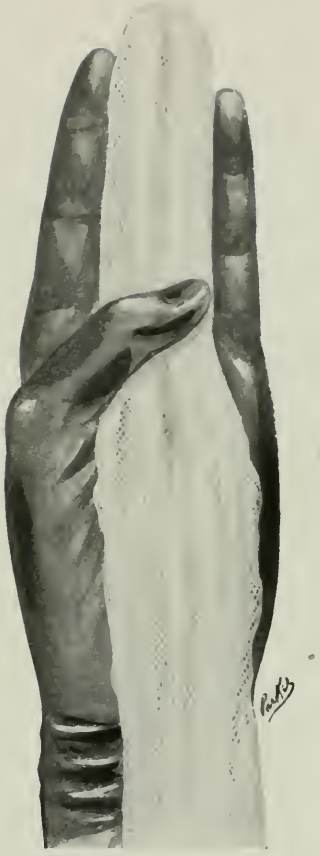
Fig. 10.

The fingers form a smooth cone, and by gentle pressure overcome the resistance of the contraction ring.

Fig. 11.

The finger works smoothly between the placenta and uterine wall, the outside hand aiding by counter pressure. The abdomen is covered by a sterile towel.

Fig. 12.



It is not easy to clean off the endometrium with the smooth rubber gloved fingers. The uterus should be wiped with the fingers covered by gauze.

Fig. 13.

Compression of the abdominal aorta by thumb and finger.

Treatment after the Placenta is out.—In all cases, as soon as the placenta has been delivered, inspect it carefully before throwing it in the drainage jar. This is done at once because if a piece of it is missing one may adopt appropriate measures, and will not be surprised by a hæmorrhage after the patient has been prepared for bed, or, indeed, after the physician is ready to leave the house. If convinced that a piece of the placenta has been retained it must be removed, for which the method has been just described.

If the membranes are missing or incomplete should you enter the uterus for their removal? Opinions of authorities differ. In maternity hospital practice, with the protection of sterile rubber gloves and an aseptic confinement room, the writer removes such retained membranes. If only a third or a half of the membranes are left, providing there is no hæmorrhage, the writer does not advise their removal. In private practice under usual conditions it is safer to trust the expulsion of the membranes to nature. Ergot and hydrastis are given for two weeks, and usually the uterus gets rid of the membranes in the first eight days.

In all cases of post-partum hæmorrhage, a brisk uterine massage is necessary. Uterine contraction has a deterrent effect even on the flow from cervical laceration. The first motions should be slow and even, but unless the uterus responds promptly they should be rapid and vigorous, spread all over the fundus and also at the sides near the tubes and ovaries. Another method recommended by Kumpf (*Cent. für Gyn.*, 1897, p. 289) is compression vibratory massage. The uterus is grasped by the right hand, pressed down into the inlet, the arm is held rigidly and then a rapid vibratory massage is administered, the excursions of the muscle being very small. If the other hand happens to be in the uterus in some other manœuver, a combined massage may be thus given. The writer has thought of trying one of the mechanical vibrators on the market, but never has had the opportunity. As an aid to uterine massage the accoucheur may irritate the sympathetic ganglia around the bifurcation of the aorta. The hand very gently rubs over this region, and uterine contractions quite often result. If the hæmorrhage is very profuse, threatening the woman's life, the same hand compresses the aorta firmly against the spine (Fig. 13). This will check the flow till the uterus has obtained control of itself, or until

the attendant can prepare the patient, his instruments, gauze, etc., for effective hæmostasis.

Ergot, a drachm of the fluid extract, is administered by mouth, and in urgent cases a hypodermic of the same is given. The best place to inject is deeply into the muscles of the thigh. The writer uses ergot aseptic, coming in little glass globules. In no instance should the accoucheur wait for the action of the drug, which may be absent or at least delayed five to thirty minutes, during which time the woman may die. Experience has proven that it is not wise to give ergot until the placenta has left the contracting portion of the uterus. The writer, therefore, does not recommend its prophylactic use in the third stage. The danger lies in the production of a spasm of the uterus which incarcerates the placenta, and sometimes brings about difficulties in removing it. The older writers speak of this danger. The disuse of ergot nowadays is so general that such cases rarely occur.

The next procedure is the hot uterine douche. The douche bag should be prepared in every labor and should be hung near the bed, properly protected against infection. The patient is brought across the bed with the hips well over the edge, *the parts are sterilized and everything arranged as for manual removal of the placenta;* the hand is inserted into the uterus and, aided by the outside hand, all clots, membranes, and shreds gently wiped out, after which the douche nozzle is introduced into the uterus and 1 quart of hot (120° F.) 1 per cent. lysol solution allowed to flow through. All air should be evacuated from the douche tube before its insertion. The danger of air embolism is emphasized by most authors. Experiments on horses, dogs, etc., have shown that immense quantities of air can be injected into them with the production of only mild and transient symptoms. In the human, however, clinical experience furnishes many examples of severe collapse and death from the admission of air to the veins.

Some authors advise cold or even iced water instead of hot for the douche. The hot water is better because it is easier to obtain than sterile cold water; it does not shock an already weakened woman, and it is just as efficient. Vinegar and lemon juice have been added to the douche, or used alone, and if one can obtain them in sterile condition they may be used. In the great majority of

Fig. 14.

Compression of the uterus in anteflexion. The abdomen is covered by a sterile towel.

Fig. 15.

Tamponade of the uterus with speculum and vulsella. The hand of an assistant should steady the fundus and note that the gauze is being inserted properly.

cases the above treatment will suffice to stop the hæmorrhage completely. If it does not, one has to do with an extensive laceration or a bad case of atony of the uterus. The laceration, of course, will have been discovered when the hand was inserted either for removal of the placenta or preliminary to the douche. The treatment of lacerations naturally occurs at this point, but we will leave the discussion till later. Should the diagnosis of severe atony be made one has to adopt other measures. Time is an element in such cases. Give the uterus a little time and it will contract, but one must not allow the bleeding to continue all the while. To stop the hæmorrhage and to give the uterus this time, compress the uterus between the two hands, as in Fig. 14. The cervix is grasped by the whole hand and folded together, while the outside hand forces the uterus down firmly against the pubis and inside hand. One may find the uterus gradually assuming its normal shape and consistency, and when, after ten or fifteen minutes the pressure is relaxed, the physician finds that the flow has ceased and he may remove his hands. Should the flow continue in spite of this manœuver, or should it recommence after he has removed his hands, no further time and blood should be wasted, but one should proceed at once to a firm tamponade of the utero-vaginal tract.

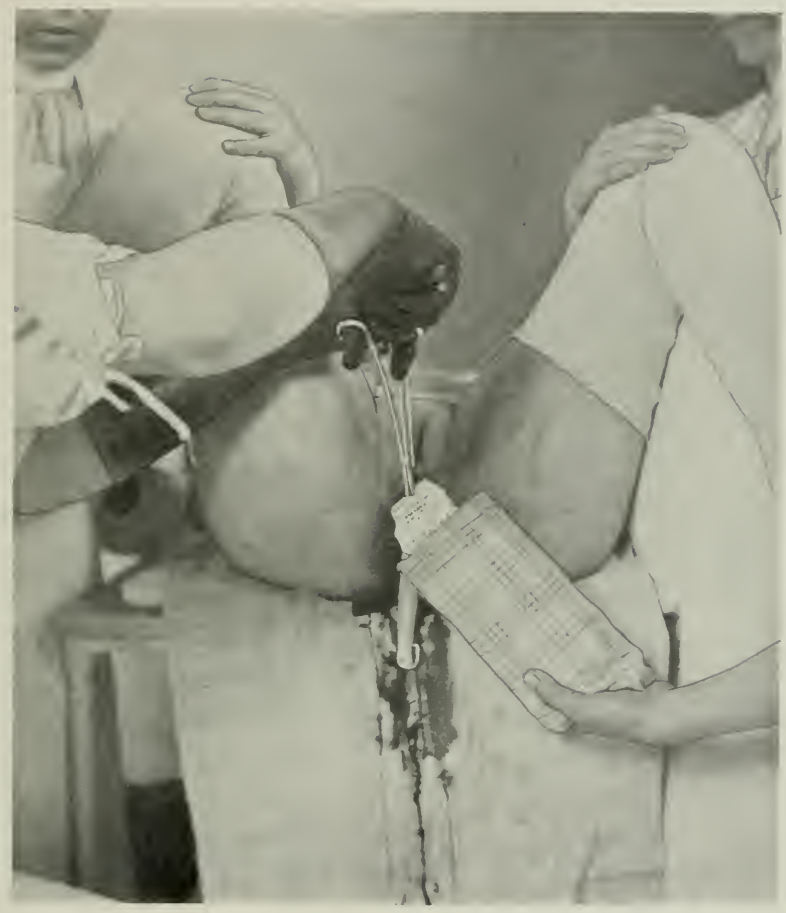
One may tampon the uterus with the hands alone, or with the single aid of the uterine packing forceps, or by sight, having drawn the uterus down with vulsellum forceps and spread the genitals by specula. If the patient is on the table and one has sufficient assistants the latter method is to be preferred (Fig. 15). A broad speculum retracts the perineum, one vulsellum forceps is placed on the anterior lip of the cervix and, if necessary, one on the posterior lip, the operator holding one, the assistant the other. The nurse holds the gauze jar near the vulva, exactly as in the illustration (Fig. 16). With the long uterine packing forceps the end of the strip is carried up to the top of the uterus, taking care that the tip of the forceps does not catch on the contraction ring or in a fold of the relaxed uterine wall. A sterile towel is laid on the belly, and with his left hand the operator determines that the tip of the forceps carrying the gauze is really in the highest point of the uterus. The gauze is then evenly packed into the uterine cavity, from side to side, and from above downward. The first part must be most firmly

placed. When the uterus is full, the forceps is laid temporarily in the basin, two fingers of the left hand are placed against the tampon, the right hand grasps the uterus through the sterile towel, and the fingers force the gauze upward more firmly, into the fundus, thus leaving room in the cervix for more gauze. This manœuver must be performed carefully and gently, and one must leave a vent for the escape of air, otherwise there is danger of air embolism (Fig. 17). After the uterus is firmly packed, the vulsella are removed and the vagina is firmly tamponed with the remaining portion of the strip. Duehrssen advised tamponing the vagina with cotton, which is better, but gives more pain and trouble in removal.

Most often it will be necessary to operate with insufficient help, and then the second method is employed (Fig. 18). The operator puts the left hand into the vagina, with the fingers inside the uterus as high as possible. The right hand, armed with the long forceps, inserts the gauze, while the fingers dispose it as it should go, into the irregular uterine cavity (Fig. 18), The inside hand acts as a grooved channel along which the gauze and forceps find easy direction. In the absence of a nurse or other persons to hold the jar of gauze near the vulva, it may be held between the knees.

The writer in several cases of great urgency applied the gauze successfully with the hands, the left hand forcing the strip up into the uterus as it was fed into it by the right hand (Fig. 19). Does the packing always stop the hæmorrhage? Yes, in all but a very few cases. Chrobak, Ahlfeld, Bumm, R. v. Braun-Fernwald, report 10 failures in over 120 cases. A more exact study of the histories of these failures shows that too much was expected of the operation. The writer has applied the uterine packing in over 75 cases. Only twice was it necessary to remove it and place a second one; twice it was necessary to sew the cervix together over the uterine tampon, and never did the gauze fail to stop the hæmorrhage. Once it was impossible to obtain enough gauze to fill the uterus, and the patient began to flow an hour afterward. The bleeding ceased with massage and the woman died of the sepsis from which she was suffering before delivery. Without doubt most of the reported failures are due to poor technic: the gauze is not put in correctly. Fig. 20 shows the parturient canal correctly packed, Fig. 21 a case where the operator has failed to grasp the situation. If the first tampon is unsuccessful

Fig. 16.



The operation of packing the uterus, showing how nurse holds jar of gauze near vulva.

Fig. 17.

Pressing the gauze into the uterus to fill out the irregular cavity evenly.

FIG. 18.

Packing the uterus, using the hand as a speculum. Method recommended The hand of the assistant steadies the uterus.

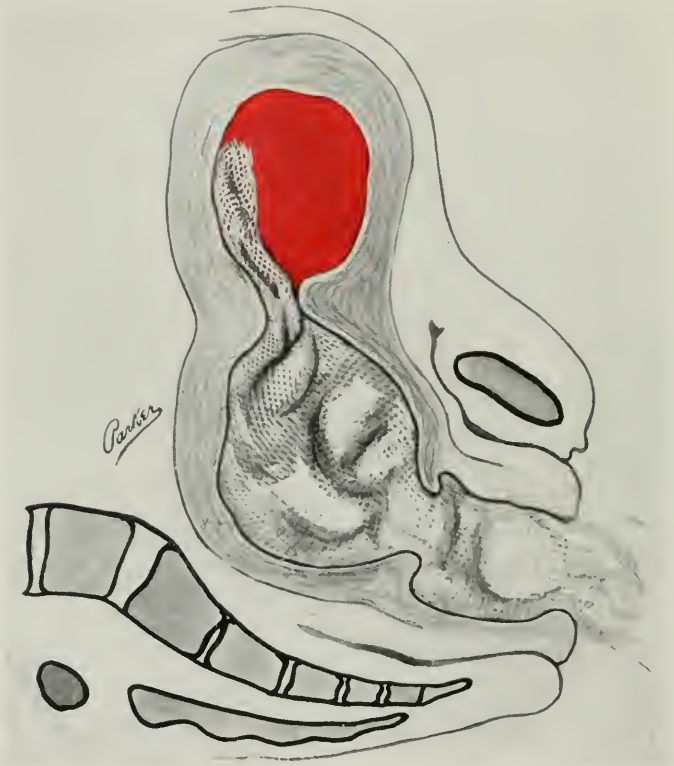
Fig. 19.

Packing the uterus with the hands alone. The belly is covered by a sterile towel.

Fig. 20.

Gauze packed into uterus correctly.

Fig. 21.



Gauze packed incorrectly. Note the uterus fills with blood above tampon.

it should be removed and another inserted,applying the last one more snugly. If absolutely sterile and non-toxic gelatine is at hand the gauze should be well saturated with it before being applied. Another way is to sew the cervix together over the tampon, as shown under "Lacerations" (Fig. 24). Some cases are so obstinate that extirpation of the uterus must be the last resort. One case of all these reported recovered. After the tamponade ergot is given as usual, and the hand should guard the uterus, massaging it lightly, or rubbing the aorta gently, if there be any tendency to further atony. A tight abdominal binder is recommended by some, but the writer has not seen any real indication for this. The gauze is removed in twenty-four to thirty-six hours, and must be withdrawn very slowly, thirty to sixty minutes being consumed in the operation. One must be prepared to pack again; but the writer has never found it necessary in cases of atony. The writer never uses iodoform gauze as recommended by Duhrssen, because of the danger of poisoning. No case of such poisoning has yet been published, but Duehrssen advises only a 5 per cent. gauze, whereas formerly he used 10 per cent. Sterile gauze may be used, but the writer prefers one slightly antiseptic, and has found that the ½ per cent. lysol gauze meets nearly all the requirements. It is my intention to make some experiments with collargol gauze. Theoretically such a gauze is ideal. Space forbids going into the history and other details of uterovaginal tamponade. Those interested are referred to a paper on the subject published by the writer in 1903 (*American Journal of Obstetrics,* vol. xlvii, No. 4).

To sum up briefly the treatment of post-partum hæmorrhage: First, if the placenta is in the uterus,—massage; failing this, expression of the placenta;—failing this, manual removal. Second, after placenta is out,—massage, ergot, hot uterine douche with cleaning of uterine cavity, compression of the uterus in anteflexion; failing all these, uterine tamponade.

Many other methods have been proposed and occasionally they will prove useful. Compression of the abdominal aorta, may be employed as a temporary expedient till one can prepare for more permanent measures. Packing the vagina with a ball of gauze, a towel, or anything sterile that is near at hand, and pressing the uterus against this from the outside, may limit the flow till the

accoucheur can procure a hot douche or gauze for packing. Squeezing a sponge with vinegar or lemon juice in the uterus has already been mentioned. These fluids must be sterile.

Fritsch recommended pulling the fundus forward over the pubis, and filling up the space between it and the promontory with towels, after which one was to put on a very tight abdominal binder. The uterus has been folded together from the side, twisted on its axis, turned inside out and ligated, etc. Chloride of iron, Monsell's solution, have been injected, and even packed in the uterus on gauze. These are unqualifiedly condemned because dangerous, harmful, useless, and unnecessary. Adrenalin has been used hypodermically and locally. Theoretically its use is correct, practically it has been used too little for final judgment.

Parsenow recommended pulling the cervix down to the vulva with vulsellum forceps, thus stretching and kinking the uterine vessels, and Henkel advised placing a bullet forceps, temporarily, on the uterine vessels at the sides of the uterus through the lateral fornices. Henkel's manœuver is not entirely safe, and has been seldom used, but Parsenow's is safe and may control the bleeding more or less till the uterus may be packed.

Treatment of Lacerations.—When a tear of the parturient canal has been discovered the treatment of the case must be modified somewhat. If you compress a clitoris, vulvar or lower vaginal tear for a few minutes, very often the bleeding will cease and the regular conduct of the third stage will not need to be altered. If compression does not suffice, a few sutures placed as in Fig. 22 will always do so. Higher vaginal tears and cervix tears, especially in placenta prævia may cause most serious hæmorrhage and place painful responsibilities on the accoucheur. It is important to get rid of the placenta at once and get contraction and retraction of the uterus. This part of the treatment is the same as for atony. If the bleeding continues, there are two courses open, one utero-vaginal tamponade, the other suture of the laceration. One may combine the two procedures. Without doubt the best way to stop hæmorrhage from a laceration is to sew up the tear, but it may be impossible to do this because of lack of proper preparations, of assistants, light, or skill. Then, too, sometimes the bleeding is so profuse that you cannot see where to place the stitches, the whole field being a lake of

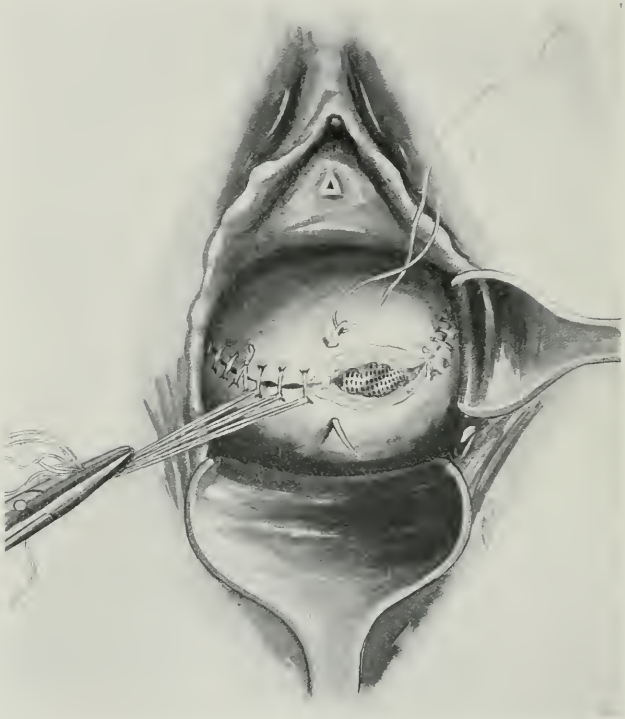
Fig. 22.

Sewing a bleeding clitoris tear.

Fig. 23.

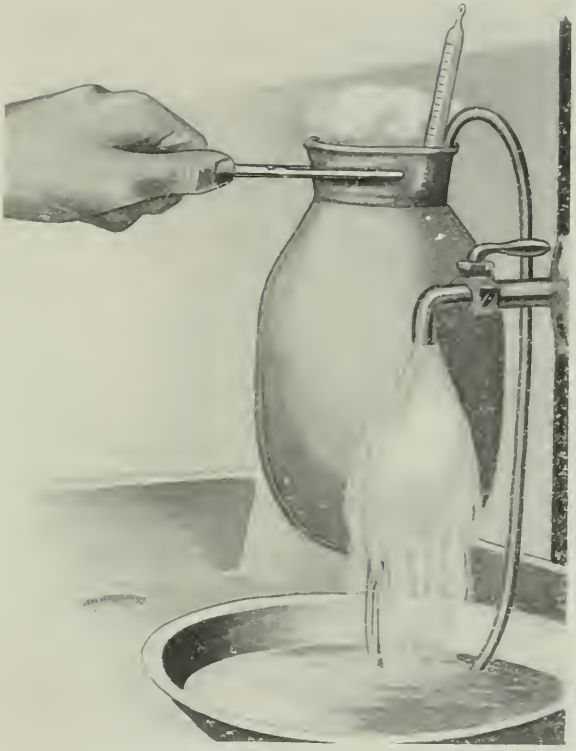
Sewing up a lacerated cervix. The two lips are drawn down with vulsellum forceps. A short curved needle is used. Since haste is imperative a continuous suture is applied.

Fig. 24.



The sides of the cervix are sewed with a continuous suture of catgut. The uterus has been packed. The lips of the cervix are now closed over the packing with interrupted sutures of silkworm gut.

FIG. 25.



Cooling the prepared salt solution under the cold water faucet.

Fig. 26.

Showing height of salt solution container and point of injection under breast.

blood. It may also be inadvisable to sew up the tear, because of sepsis or because the facilities are not at hand for aseptic work. It may thus be safer to leave the wound open for drainage. In such cases the tampon will be indicated, but it is not so certain a remedy as in atony of the uterus. A hæmorrhage from deep cervical tear is a formidable accident, and in placenta prævia even a superficial tear may be fatal.

In packing a uterus and tear, one must be careful not to extend the latter. This may be directly fatal. The uterus and vagina should be firmly tamponed, the tear smoothly and snugly tamponed, but the parts must not be handled roughly.

From past experience with cervix and lower uterine segment tears, the writer has the deepest respect for these structures and avoids lacerating them at all hazards. Thus he has been spared, of late years, the painful situations that formerly developed from performing rapid extraction of the fœtus through an unprepared parturient canal. The greatest field, then, in the treatment of lacerations is prevention.

A simple lateral cervix tear is repaired as in Fig. 23. A broad speculum retracts the vagina, the cervix is drawn into reach by double vulsellum forceps, and the edges of the tear approximated by suture. The first stitch should be placed well up in the fornix. If the tear is inside the cervix, extending into the broad ligament, it will be necessary to open the cervix by separating the forceps and sewing from the inside. As the upper angle of the wound may be out of reach, the lower sutures are placed first and, drawing on these, successively higher portions of the tear may be closed. An assistant's hand may force the uterus down from the outside. It is this latter class of cases in which the field is inundated with blood, and in which it is so very necessary to see. One may pack one side of the uterus while sewing the other, or pack the uterus tightly and sew the cervix together over it (Fig. 24). It may be necessary, because of the furious bleeding, to pack the tear with a large wad of cotton, press this firmly against the wall of the pelvis and then force the uterus down from the outside. The writer held a hæmorrhage thus in check for twenty minutes, then the vagina was lightly packed and the suture of the rent postponed till the next day. The operation saved the woman's life.

Henkel's method of clamping the bases of the broad ligament with bullet forceps would be most useful here, and in some cases may be performed. The uterus is pulled well down and to one side. The dangers are perforation of the vessels of the ureter and opening the peritoneal cavity, but where the bleeding defies the tampon and suture, it should be tried. By keeping close to the uterine wall most of the dangers are avoidable. It was tried in 19 cases in Olshausen's clinic; in 17 cases the bleeding was stopped, but 5 women died from loss of blood, which means that the remedy was applied too late. I have never used it, but will retain it for such an emergency.

AFTER-TREATMENT

All danger is not past when the hæmorrhage has been stopped. The patient may die from the loss of blood. Unless one has plenty of medical assistants, this part of the treatment will have to be left till the accoucheur has stopped the active hæmorrhage. This is the advantage of doing obstetrics in a maternity,—while stopping the loss of blood an interne may be filling the patient's blood vessels with salt solution.

During obstetrical operations in general, and the treatment of hæmorrhage in particular, the patient should be kept covered and warm. After a hæmorrhage has been checked the patient should be warmly covered in bed and surrounded by hot water bottles. If she is in much shock the foot of the bed should be elevated at least two feet, and a hypodermic injection of camphorated oil given. To tide the patient over a fainting spell, give a hypodermic of aromatic spirits of ammonia, and let her smell some smelling salts. While doing these things saline solution for subcutaneous administration should be prepared. Sterile salt in 2-drachm vials should be carried in the obstetrical satchel. The douche bag, a bath thermometer, and needle should be boiled. Two drachms of this sterile salt are placed in the bag; the tube, armed with the needle, is also put in the bag, then 1 quart of boiling water poured on the salt. The mouth of the bag is stopped with a large wad of sterile cotton and the bag is held under the cold water tap or in a dishpan of cold water, till the thermometer registers 115° F. (Fig. 25). The skin under the breasts is prepared aseptically, and the needle, with the solution flowing, is put in, below the mammary

seeks to direct the needle into the connective tissue under the glandular portion of the breast. The bag is now raised as in Fig. 26. As much as 32 ounces may run under the skin. It is advisable to lower the bag occasionally to prevent over-stretching and too rapid opening of the lymph spaces. If the case is critical the other side may be injected, as well as the two flanks. In one patient I injected 116 ounces of saline solution in one sitting, using the four points named. The patient recovered. This patient also absorbed 2 quarts from the rectum. Shall you inject the salt solution while the hæmorrhage is still going on? Yes, subcutaneously, not intravenously. The blood lost then will not be pure, but mixed with salt solution, and it is not so precious. By the time a quart has been absorbed the accoucheur must have the hæmorrhage in his control or the patient will have died. Some authors advise against the use of saline solution till after the flow has ceased. I think their objections are theoretical. Even in cases of intra-abdominal hæmorrhage, while preparing to open the belly, I would administer saline solution subcutaneously.

The writer has never seen fit to bandage the limbs, so-called autotransfusion. The position of the body with the foot of the bed elevated is sufficient, aided by saline solution. Without doubt salt solution has saved many lives. It is not wise to administer it per rectum until the hæmorrhage has been permanently stopped, because if any local treatment is to be carried out, the discharges from the bowel may infect the field, the hands, instruments, gauze, etc., so that sepsis may be the direct result. One of the most unwelcome incidents in any obstetrical operation is a flow of liquid fæces from the anus, and the writer is certain that it has caused infection and death many times.

By mouth the patient may have wine, hot coffee, water, or a hot oyster stew. If the patient vomits she usually feels relieved, after which liquids may again be given.

During the first week nourishment must be given freely. The bed is lowered in two or three days or sooner, depending on the condition of the patient. It is lowered a few inches at a time to avoid syncope.

Not seldom after severe hæmorrhages the puerpera will have a rise of temperature to 102° F. within the first twenty-four hours.

This is reaction fever and will go down without treatment. But not seldom sepsis follows in the wake of a severe loss, first because the resistance of the patient is so lowered that infection finds easy access, and second because in the excitement and unpreparedness attending most of such cases the rules of asepsis are forgotten or impossible of being carried out in detail. Sepsis under these conditions is likely to follow a severe course.

The milk secretion is diminished, in spite of Ahlfeld's statistics. Ahlfeld had strong peasant women to deal with. The high-strung American mother cannot stand the drain of lactation after severe loss of blood. My experience has been unequivocal on this point, and I notice that v. Braun warns against forcing the nursing of the infant on neurasthenic women. One may permit a partial breast feeding until the woman has completely recovered from the exhaustion of the hæmorrhage. Some women recover very slowly from the spanæmia and require blood tonics for long periods. The best treatment the writer knows for such cases of severe hæmorrhage is living at the seashore, and the woman should be sent there as soon as she can bear the journey.

APPENDICITIS IN PREGNANCY, WITH AN ACCOUNT OF SIX CASES

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Case I.—Is that of a lady whom I attended in her two confinements in the years 1898 and 1901. The history she gave me was that in 1893 and 1894 she had been treated for appendicitis.

The symptoms on which the diagnosis was established were "high fever, pain in the lower abdomen, sickness, and thickening detected on pressure in the right groin." This was noticeable for a considerable time afterwards. The patient was in bed for two or three weeks with each attack. The second was the more severe; the temperature rose to 105° and the vomiting was very distressing; this was in the year 1894. About two years later the lady married, and fell pregnant in January, 1898. In the intervening years since the last attack there had been "occasional slight attacks of pain and discomfort." On examination in the fourth month of gestation I could not find any thickening, nor elicit any tenderness in the region of the appendix. The patient's health improved remarkably as gestation advanced. The puerperium was afebrile and the appendix gave no trouble.

During the second pregnancy the patient's health was not good and constipation became very troublesome, but still the appendix showed no sign of inflammatory reaction. The labor, which occurred on January 1, 1901, was normal, as was also the puerperium, and up to the present, February, 1906, there has been no recurrence.

In this case the first pregnancy seems to have had a beneficent effect on the appendix-inflammation—the latter caused occasional attacks of pain until the patient became gravid, since when it has been quiescent, if not cured entirely.

Case II.—In the second case the lady was more or less a chronic invalid; there was a family history of pulmonary tuber-

culosis, of which disease, however, the patient herself showed no sign.

The first confinement took place on February 15, 1895. Throughout the whole gestation period the patient suffered from constipation and flatulent dyspepsia. Labor and puerperium were normal.

During the second pregnancy there was pain in the right iliac fossa, accompanied with sickness and constipation, but this was put down to gestation molimina. The labor was normal, but in the second week of the puerperium acute abdominal symptoms set in; the abdomen suddenly became "blown out like a balloon." There was high fever, and great tenderness over McBurney's spot; the accoucheur diagnosed appendicitis. This attack subsided under treatment and the lady continued to nurse the child. The periods appeared one month after the confinement and the flow lasted for three weeks; it ceased for fourteen days and then came on again, lasting this time for four days. After this, menstruation became regular, but scanty, coming on every month and lasting four days, three diapers being used at each epoch. The patient ceased nursing when the child was five months old, on account of diffuse alopecia and general debility. In December, 1904, I saw this patient in consultation. She was then in bed, suffering great pain in the lower abdomen; constipation was very obstinate and her temperature was 101° F. I made the following notes: "Aged 38 years, married 13 years, two children, youngest 3½ years. There have been repeated attacks of right iliac pain, as many as three or four every year for the last ten years. These attacks have come on suddenly and are accompanied with nausea, but never with vomiting; they have been more frequent during the last 18 months. The present attack (December, 1904) began a fortnight ago with sudden pain in the right iliac fossa and groin, the pain ascending to the colon. This was in a measure relieved by a turpentine enema, which brought away a very copious hard stool. The menstruation has been regular, but scanty; 3 days' loss every month." On examination I found the lower abdomen slightly distended, and universal tenderness over the right iliac and hypogastric regions, but there was no area of dulness and no tumor was palpable by the abdomen. On examination by the vagina, a cystic swelling was found high

up on the right side of the uterus. It was exquisitely tender, and fixed. I regarded it as a tubal or ovarian collection of fluid, adherent to cæcum or appendix. I kept the patient in bed another fortnight, and at rest for two months from the onset of the attack. The general practitioner was in constant attendance and had great difficulty in combating constipation, which continued until March, 1905, when, although considerably improved in health, the patient consulted me again. On this occasion I elicited the fact that "another attack had threatened, but had been aborted by a turpentine enema." I made the diagnosis of right-sided salpingo-öophoritis with indefinite swelling in the appendix region, and advised exploration. This was carried out on March 16, 1905. The distended cæcum overlay the uterus and right appendages in the true pelvis. The right tube was adherent in its outer third to the dependent appendix vermiformis. The right ovary was cystic and of the size of a tangerine orange. The cæcum was adherent to the fundus uteri by fibrous adhesions. The outer two-thirds of the Fallopian tube, together with the diseased ovary, was removed, and then the appendix was amputated close to its attachment to the cæcum, the wound being closed by a mesenteric graft. The patient made a good recovery. She still suffers from constipation, but is otherwise well and "feels as different as possible to what she used to do."

Case III.—The third case is a very sad one. The lady was thirty-eight years of age at the time of her first and only attack of appendicitis. On June 23, 1901, I explored for an ill-defined pelvic swelling and found the left ovary adherent to the sigmoid flexure by inflammatory bands; there was a cornual fibroid, which I did not remove. The right adnexa were healthy and the appendix vermiformis was not seen. The patient made a good recovery and married a year later. On August 1, 1903, I attended this patient in her confinement, which was normal, as was also the puerperium. There had been marked constipation during gestation, but this, together with dyspepsia, had been troublesome for many years before.

On December 11, 1904, the patient was seized suddenly with violent umbilical pain during a meal; on leaving the dinner-table she became violently sick and passed two or three large stools.

The temperature on the same evening dropped to 95.8° On December 13 the temperature rose to 100.2° and the pulse to 94. The pain shifted into the left iliac region; both recti were very tense, and respirations became rapid, *i.e.*, 32 per minute. The urine drawn off by catheter was deep orange in color; sp. gr., 1030; it contained no albumin. At 5.30 P.M. the temperature was 100.2°, pulse 96, respirations 30. I arrived at 6 P.M. and found the patient had just vomited a bilious yellow fluid. The pulse was 112, the temperature 100.2°. The abdomen was quite rigid (there was no abdominal breathing), it was uniformly tympanitic and very tender in the right iliac and groin region below McBurney's point. The uterus was fixed but not displaced; there was great tenderness in Douglas' pouch, but no tumor was discovered; the external os was slightly patulous, the cervix soft and velvety. Per rectum a small body the size of the ovary, but exquisitely tender, was felt in the right posterior quarter; the patient shrieked with pain when I touched it, and ⅛ gr. of morphia was given subcutaneously to allay the distress caused by vaginal and rectal examination. Immediate operation was decided upon, having previously committed to writing in my notes: "early extra-uterine abortion or acute appendicitis with perforation." Directly a median incision through the anterior abdominal wall was made, there was an escape of foul gas and flocculent turbid fluid. The intestines were much distended, the lower coils intensely injected. On passing the hand into the pelvis the appendix came into view without traction. It was elongated, thickened and gangrenous. Hanging from the edge of the hole which admitted my index finger, was a stercorolith, the size of a hazel nut. The entire pelvis was filled with pus; there was no attempt at isolation of the infected area by adhesion. The uterus and adnexa were uniformly covered with a thick coating of yellow lymph. The fundus uteri was enlarged. The treatment consisted in carefully sponging out all the fæcal pus, sponging the coils of intestine one by one, amputating the appendix and thoroughly irrigating the intestine with three drachms of sulphate of magnesia in solution. The abdominal wall could not be brought together, so no attempt was made to compress the intestines; but they were overlaid with gauze, and fishing gut sutures passed through, and clamped on either side of the wound,

ready to tighten up and tie as the swelling subsided. The patient's condition seemed to improve during the next day (December 15), and an opening was made in the pouch of Douglas and a further collection of pus escaped. On December 16 violent black vomiting set in and the patient succumbed at 8 A.M.

An examination of the appendix and uterus showed that the latter presented a very swollen tubercular-looking endomentrium, and on the apex of one of the tuberosities there was a crescentic spot, like a cicatrix. This was carefully drawn in color as soon as the uterus was opened. The part was then excised, fixed, hardened, embedded in paraffin, and from it I have obtained between three and four hundred serial sections. They prove the presence of a very early intact ovum, about which I shall have more to say elsewhere.

The appendix measured five inches in length; it was much thickened, having a diameter near the apex of one inch. It was bent upon itself at the mid-point in its length, the flexure making an angle of 120° on the attached or mesenteric side. At the apex of this bend there was a large perforation. The latter measured 1 × ½ inches. Attached to the lower end of this hole was a stercorolith the size of a filbert nut. The whole organ was of a chocolate-blue color with patches of greenish lymph adherent to it. The mesentery was thickened by congestion and œdema.

Case IV.—For the details of this case I am indebted to my friend, Dr. Gibson, of Porchester Terrace. The patient married in 1899. On April 5, 1903, when eight months gravid with her third child, she was seized at midnight with violent sickness, abdominal pain and rectal tenesmus. This was put down to partaking of German wurst for supper. The attack subsided by the morning without any acceleration of pulse or rise of temperature. Sulphate of soda was given, and on April 7 the patient seemed well enough to get up from bed. Hot milk was given before she attempted to rise, and this was followed by violent abdominal pain, which lasted an hour. A quarter of a grain of morphia was given on the evening of this day, as the pain returned, and the patient slept until 6 o'clock the next morning (April 8). She then got up to micturate and, after one sharp pain, the baby was born; this was during the eighth month of gestation. After the labor was over the tempera-

ture rose for the first time during the illness—it then registered 100° F. Diarrhœa set in, the motions were very fœtid, and paroxysmal pain commenced and lasted three or four days. Castor oil and laudanum were administered. A tumor now developed. It was situated above the level of the anterior superior iliac spine, high up in the loin; it was thought to be too high for the appendix, and fæcal accumulation was diagnosed. For the first three days of the puerperium the temperature ranged between 99° and 100°. The patient's condition improved sufficiently to let her get up on the seventeenth day after labor, but when she had been up for one hour the pain came on again. A physician was called in consultation and it was decided that the appendix was free, but that the case was one of perityphilitis. Sulpho-carbolate of soda was given internally and poultices applied. Four days later the patient again essayed to get up, but soon was obliged to return to bed for another fortnight. She was now seen by a surgeon, who concurred in the diagnosis of perityphilitis and decided not to operate. The patient was kept in bed until May 23. She got up, but on May 24 she had the worst attack of all and the temperature rose to 103° F. and the pulse rate was 120 per minute. A consultation between a physician, surgeon and accoucheur led to the decision to explore the abdomen. On May 31 this was done. The cæcum and ileum were matted together, between them about two drachms of pus was found, and also what was taken to be the stump of a necrosed appendix, which lay at the bottom of the cavity from which the pus escaped. This cavity was drained with gauze brought out through the abdominal wound. For the first week all went well, when the pain started again, and the temperature rose to 100° F. Still the wound looked healthy. Then three weeks after the operation another severe attack occurred, the temperature rising to 102° F. This lasted for twenty-four hours, during which time the surgeon removed a pessary which the obstetrician had inserted for retroversio uteri. In the middle of July, the patient undertook a long train journey, but she could not walk more than a few yards at this time. On August 12 another relapse set in, the temperature rising to 102° F., as before. There was marked sickness and abdominal pain; there was no special tenderness over the abdominal scar; there was fœtid diarrhœa. This attack lasted ten days. Still another bout of the symptoms appeared

on September 14, and now a mass, the size of a duck's egg, was discovered in the left fornix. The patient was kept in bed for six weeks with hot douchings and tamponading; the skin of the abdomen was painted with iodine. Under treatment the pain subsided, and in November the patient traveled from Scotland to London. There was a relapse immediately afterwards, but a London gynæcologist could find nothing abnormal in the pelvis, except slight backward displacement of the uterus. The patient was therefore allowed to get up, and she returned to Scotland on December 17. She had another attack in the train and arrived at her destination very ill indeed. The surgeon who did the first operation explored again, on January 11, 1904. The ileum and cæcum were again adherent; the appendix was found surrounded by a small quantity of pus, whilst adherent to it was a right-sided hydrosalpinx. Since this operation the patient has been perfectly well. It is noteworthy that in this case there had been no constipation, nor was there any indication that the appendix was diseased until three days before the premature birth of the child. The child is alive and well.

Case V.—I am indebted to Mr. Marmaduke Sheild for the following details and for permission to publish them.

In the year 1891 Mr. Sheild was called in consultation to see Mrs. X., aged 38, multipara, the subject of recurrent attacks of appendicitis. The patient was taken suddenly ill with acute abdominal pain, vomiting and high temperature, during the seventh month of gestation. She had all the signs of acute general peritonitis when Mr. Sheild saw her. On opening the abdomen he found a gangrenous appendix lying free in the peritoneal cavity; there was no abscess formation, but acute generalized suppurative peritonitis existed. Dr. Routh, who was present, dilated the cervix and delivered a seven months' still-born child. The peritoneal cavity was flushed and drained, but the patient died shortly after the operation.

Case VI.—Mr. Marmaduke Sheild has also kindly allowed me to mention this case, which he saw in consultation with a London practitioner in 1895. The patient was aged 23 years, she was a primipara, and was three months pregnant when obscure right-sided pains, unaccompanied by sickness, set in. There was no history of previous attacks of appendicitis and no palpable tumor.

The uterus corresponded in size to a third month's gestation; nothing abnormal was detected in the pelvis. There was tenderness and resistance over the right iliac fossa, and this, having regard to the former case, induced Mr. Sheild to advise an exploratory operation. He found the appendix adherent to the parts around and kinked upon itself; the uterus lay below the pelvic brim; the appendix was in the abdomen and not adherent to the right adnexa, which were still within the true pelvis. The appendix was removed and the patient made a good recovery, the pregnancy going to term and ending in a normal labor and puerperium.

The relationship of appendicitis to pregnancy forms one of the most important and, at the same time, one of the most difficult questions for obstetricians and gynæcologists. The literature on the subject is as yet too scanty for us to draw accurate conclusions as to the frequency with which appendicitis complicates pregnancy, labor and the puerperium. It is highly probable that the appendix is accountable for many of the symptoms, such as pelvic pain and vomiting, which are attributed to the molimina of pregnancy; in other words it is more than likely that slight attacks of appendicitis during pregnancy are overlooked and explained away, whilst only the more severe cases are diagnosed and put on record. This opinion gains ground when we remember that the period of fertility corresponds with that in which appendicitis is most commonly met with.

The six questions which naturally present themselves in connection with this subject are:

(1) Does pregnancy predispose to appendicitis?
(2) How does pregnancy influence this disease?
(3) How does appendicitis affect pregnancy?
(4) How does labor affect appendicitis?
(5) How does appendicitis affect the puerperium?
(6) What is the prognosis for the child and the mother?

If I were to attempt to answer the first question from my own limited clinical observations I should find my facts conflicting, for in Case I the reminders in the shape of "occasional attacks of pain" all passed away during the first pregnancy and never returned; whilst in Case II it was different. Here a definite attack occurred during the second puerperium, the patient being the subject of recurrent appendicitis, which had extended over a period of ten

years. In Case III it is practically certain that the conception, which was only of a few days' duration, had nothing to do with the perforation of a previously diseased appendix. In Case IV the question becomes more difficult. There had been no constipation, nor any previous intimation until the eighth month of gestation that the appendix was at fault, whilst at the operation this organ was found to be adherent to the right Fallopian tube. There had been two normal labors previously, and the fact that in the third the appendix became diseased seems to me to be a coincidence and purely accidental. Writers on the subject differ extensively in their opinion as to the etiological relationship between pregnancy and appendicitis. Boije, taking for granted that appendicitis is rare in pregnancy, argues from this that the latter cannot be a predisposing cause of the former; again, the disease is slightly more common in men, which we should not expect if women incurred an additional risk in becoming pregnant. Kelly says it is not necessary to assume a direct etiological relationship to account for the recurrence of appendicitis during pregnancy, and probably in the majority of cases the coincidence is purely accidental. E. Fraenkel is equally or even more concise in his statement, for he says that "such a movable organ can easily become adapted to the varying conditions of pressure caused by pregnancy in the abdomen," adding "that if pregnancy predisposed to it, appendicitis would be as common during gestation as it is, fortunately, rare." Again, Winckel in his Handbüch für Geburts, Bd. II., Tril II., S. 1424, says, rather emphatically: "Pregnancy, as such, does not predispose to appendicitis."

In answer to the question how does pregnancy influence appendicitis? it is most generally agreed that pregnancy may exert a directly unfavorable influence upon an old or recently inflamed appendix. This is more likely to occur after the fourth month of gestation, as it is then that the uterus first rises up into the false pelvis, and begins to open up the layers of the broad ligament, with consequent engorgement of the ovarian veins. The fundus uteri, as Füth has pointed out, may now lie in close apposition to the cæcum, and the appendix may lie in close contact with the right appendages. My second case shows what may happen under such conditions. We saw that the cæcum was adherent to the top of the

uterus and the appendix adherent to the right tube. In this particular case no urgent symptoms occurred until the end of the involution period, when the cæcum and appendix had been dragged down into the small pelvis. Hlawecek is of opinion that pregnancy will invariably cause an exacerbation of a recent, or light up an old, not completely healed inflammation of the appendix. He says that the appendix may be unfavorably placed from the point of view of its own nutrition, and argues that the growth of the uterus occasions traction on the inflamed organ, and this view is quite in accordance with Füth's researches upon His's models; but we must bear in mind that the pregnancy does not set up the predisposition to the disease. Only when the appendix is inflamed will its close relationship to the gravid uterus give rise to trouble. The fact that *severe* appendicitis is rare during pregnancy is explained by Boije as due to the cellular hyperplasia and increased blood supply to the pelvic organs during gestation. Krönig says that the hyperæmia is not confined to the pelvic organs, but is shared by the serous tissue of the peritoneal cavity generally, and also by adhesions. This increased circulation favors absorption, and thereby exerts a beneficial therapeutical influence upon the inflamed tissues, acting in the same way as does the hot air treatment. The influence of pregnancy on chronic inflammatory processes has never been studied, and as Füth says, it is not easy to prove its effect, since chronic pelvic lesions militate against the occurrence of pregnancy. He adds, however: "In the cellular hyperplasia of pregnancy is to be seen a reason for the rarity of *severe* appendicitis during gestation." He publishes a case which proves that repeated severe attacks, late in gestation, need not lead to serious results. This, however, is probably an exception to the rule. The first case, however, in my series goes to prove that Hlawecek's statement, that pregnancy invariably lights up an old attack of appendicitis, is too sweeping, for here, as already stated, the attacks of pain subsided during the first pregnancy.

Our third question, as to how appendicitis affects pregnancy, is answered by stating that it depends entirely upon the gravity of the lesion. In mild cases the pregnancy is undisturbed. In severe cases abortion or miscarriage generally ensues. The act of labor is accompanied by great danger in cases of acute appendicitis. The

contractions of the uterus will stretch and tear any adhesions which may bind it to the wall of an abscess, and this will result in the leakage of pus into the general peritoneal cavity. During the puerperium there may sometimes be witnessed a type of puerperal fever that is due to appendicitis. Bacterium coli has been found in the lochia, uterine wall and fœtus. Such cases have been recorded by Eisenhardt, Winckel and Krönig. Again, from the dragging of the diseased tissues into the small pelvis, a pelvic abscess may result. This has occurred after emptying the uterus of a dead fœtus in cases of appendicitis. In Case V. death followed, after operation, as the result of general peritonitis from perforation of the appendix in the seventh month of gestation. Premature labor had started before operation. Mr. Sheild's view that this patient's life might have been saved if the operation had been performed earlier, is well justified, for Kelly quotes T. K. Holmes' case, where a woman who was four months pregnant was operated upon for a gangrenous appendicitis, with general peritonitis. Recovery followed, and pregnancy went to term, when a healthy child was born. In Mr. Sheild's case there was no attempt at abscess formation, and it seems probable that the diseased appendix was adherent to the uterus or appendages, and that the crisis arose through the onset of premature labor breaking down the protective adhesions. Fraenkel, however, does not regard abortion as a cause of the fatal termination in such cases as this, but says the severe infection caused the abortion, and also the death of the mother.

With regard to the prognosis of child and mother, severe appendicitis must be looked upon as a very fatal lesion. In thirty-one cases operated upon for appendicitis during pregnancy, in eighteen abortion, with death of the child, occurred, *i.e.,* in 58 per cent. As regards the mother, in thirty-one cases which were operated upon fourteen died, *i.e.,* 45.1 per cent., and in twenty-four cases not operated upon, six died, *i.e.,* 25 per cent. (Boije.) All authors agree that acute appendicitis occurring in pregnancy is extremely dangerous for the mother and child. Hlawecek says the occurrence of diffuse peritonitis is favored by labor pains, which break down adhesions. Fraenkel, as above stated, thinks the danger is overestimated, and so does Brandt. With regard to the maternal prognosis, it is to be remembered that in pregnant women, as well as

non-pregnant women, adhesions, bands and cords may lead to incarceration and strangulation of the bowel.

The diagnosis of a typical attack of acute appendicitis will be based upon such symptoms as were met with in Cases III and IV, *i.e.,* the *sudden* onset of severe abdominal pain, accompanied by violent sickness, rectal tenesmus and general constitutional disturbance; but difficulties arise if the pain is not definitely localized, or if, as in some cases, it is referred to the left iliac fossa, or is only felt at the umbilicus. In primiparæ the onset of appendicitis is often put down to a miscarriage, and Case III shows that extra-uterine gestation has to be excluded. In the later months of pregnancy, palpation and percussion become increasingly unsatisfactory, owing to the presence of the pregnant uterus. It is advisable to turn the patient on her left side whilst carrying out the investigation. During the puerperium acute constitutional disturbances, occasioned by the rupture of an appendiceal abscess, may be attributed to puerperal uterine infection. Again, the uterus may become secondarily infected from a diseased appendix, and the foulness of the lochia resulting in consequence, together with the subinvolution, may mask the primary lesion. An abscess in the pouch of Douglas, due to perforative appendicitis, may be wrongly attributed to primary uterine and tubal infection; and in the same way, right-sided parametritis, arising from the spread of a retrocolic appendicitis, may be put down to *ordinary* puerperal infection. Great confusion arises also in deciding the true nature of more chronic cases, where there is a localized tumor to be felt in the right fornix, either early in pregnancy or in the late puerperium. Such tumors are often proved by operation to consist of a diseased appendix, adherent to the right tube, and perhaps to the right ovary. Clinically, however, this condition has been again and again attributed solely to tubal or ovarian disease, and the appendix left entirely out of consideration. If we meet with definite right-sided pain, coming on in definite attacks during pregnancy, and especially in a dyspeptic and constipated subject, we ought to keep the possibility of appendicitis clearly in view. Tubal disease as a primary lesion is so frequently bilateral that a unilateral (right-sided) pain is far more likely to point to an appendiceal lesion than to tubal mischief.

As regards the treatment of appendicitis in pregnancy, I would

say at once: Ignore the pregnancy. Appendicitis is a surgical lesion, and as such it should be dealt with on its merits. I am aware that this advice is counter to that given by many authorities.

American and French surgeons state that the indication for operation is more urgent in pregnancy than in the non-gravid state. McArthur and Marx advise operation and induction of labor. They argue that the fœtus will surely be dead and the mother is certainly in danger. Thus they advise laparotomy for an appendix abscess, and before closing the abdominal wall say that the uterus should be emptied. Marx does accouchement forcé before performing laparotomy. It is, however, very fortunate that this advice has not been followed, since pregnancy is not (as Boije has proved) necessarily disturbed by operation, and it is possible to save the life of the child. Accouchement forcé is distinctly dangerous. Boije says it is better, in operating, only to regard the pregnancy to the extent of being cautious in abdominal manipulation, in order not to set up labor pains. Hergott would have us operate on all cases, however slight, as a purely prophylactic measure, but Boije's statistics clearly show that the risk of recurrence in consequence of pregnancy has been over-estimated.

Should an abscess form in the pouch of Douglas, it should be opened per vaginam and drained. These cases do better than those where the collection of pus is situated higher up.

The point I would insist upon is, whenever an abscess forms, be content simply to open and drain, and leave the uterus alone if labor has not already started. If the appendiceal trouble is first diagnosed during labor, the latter should be first concluded, and the appendix dealt with afterwards. If appendicitis complicates the puerperium we are face to face with a very grave condition. When pus is present, open, and secure the freest drainage possible, using a counter opening through the pouch of Douglas if necessary. Should the uterus be secondarily infected, it should be removed.

THE COMPARATIVE PHYSIOLOGY OF MENSTRUATION AND THE ALLIED PROCESSES

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The nature of the connection between the changes which occur in the nonpregnant uterus and those which take place in the ovaries during the menstrual cycle is a question which is still open to discussion. There are some who hold the view, put forward by Sigismund and adopted by His, that the degeneration stage of menstruation is of the nature of an undoing of a preparation (represented by the previous growth stage) for a fertilized ovum, and that the destruction takes place because such an ovum is not present, either through the nonoccurrence of ovulation or of coition. As Powers expressed it in his well known dictum, " Women menstruate because they do not conceive." It is obvious that according to this theory ovulation must take place prior to menstruation. A modification of this view has been put forward by Beard, who assumes that the whole of the phenomena of menstruation represents a process which is of the nature of an " abortion " of something prepared for an egg given off at or after the close of the preceding menstruation and that it takes place because this egg has escaped fertilization. A third hypothesis which has gained many adherents affirms that the entire menstrual process, including not merely the growth stage but also the destruction and recuperation stages, is of the nature of a preparation for an embryo. On this theory, according to Geddes and Thomson, " the process is viewed as a kind of surgical freshening of the uterus for the reception of the ovum, whereby the latter during the healing process can be attached safely to the uterine wall." If this view be accepted there would appear to be no theoretical reason for the conclusion that ovulation must precede menstruation.

In the absence of direct evidence, which is very difficult to obtain, it is possible to throw much light on the phenomena of men-

struation and the allied processes by a study of the corresponding phenomena in the lower mammals. There can be practically no doubt as to the homology between the œstrous cycle of the lower mammals and the menstrual cycle of the primates,—*i. e.*, of monkeys and the human female. This is shown by a histological comparison of the changes undergone by the uterus and external generative organs during the successive periods of the œstrous and menstrual cycles, which present an essential similarity but differ mainly in the greater severity of the destruction of uterine tissue in menstruation.

The changes which occur during the menstrual cycle have been grouped by Milnes Marshall, Heape, and others in four periods, as follows: (1) Period of rest, (2) period of growth, (3) period of destruction, and (4) period of recuperation. The œstrous cycle of a typical lower mammal such as the bitch has been divided by Heape into: (1) Period of Anœstrum or resting stage, (2) period of proœstrum, which contains a growth stage during which the uterus and generative organs become congested, followed by a destruction stage in which blood is usually poured out to a greater or less extent; (3) period of œstrus or period of desire, at which (and only at which) coition normally occurs, and (4) in the absence of pregnancy a short period of metœstrum during which the genital organs resume the normal condition. Thus the " period of heat " in a lower mammal consists in reality of two sub-periods, the proœstrum and œstrus, and it is the proœstrum alone and not the entire " heat period " which is homologous with menstruation in the human female. This was first pointed out by Heape (1900).[1]

The correspondence between the œstrous and menstrual cycles may be indicated thus:

Period of rest		anœstrum
Period of growth...	proœstrum (Menstruation)	period of " heat."
Period of destruction		
Period of recuperation	œstrus	
	metœstrum.	

It is thus seen that the normal time for coition in the lower

[1] The histological changes which the uterus undergoes during the œstrous cycle have been worked out in the sheep, the ferret (Marshall 1903, 1904), and the bitch (Marshall and Jolly, 1905). These should be compared with the corresponding changes in the monkey during menstruation (Heape, 1894, 1897).

mammals is synchronous with the commencement of the period of recuperation.

Ovulation occurs in the dog during œstrus, or at any rate not until a late stage in the proœstrum. (Marshall and Jolly, 1905.)In the rabbit it does not take place without the stimulus of coition (Heape, 1900) and there is evidence that the same is the case with the ferret and occasionally also with the sheep. (Marshall, 1903, 1904.) It follows that with rabbit and ferret (at any rate normally) ovulation takes place during œstrus. It is evident, therefore, that the degeneration stage of the proœstrum and consequently also of menstruation, cannot be of the nature of an undoing of a preparation (represented by the preceding growth stage) for a fertilized ovum, since the normal time for ovulation and fertilization in a lower mammal is after the proœstrum is over. Consequently Sigismund's theory of menstruation is untenable. And since "heat" occurs usually in the bitch only at periods of six months, while in certain other mammals its occurrence is at even less frequent intervals, it is obvious that the degeneration stage of the proœstrum cannot represent an abortion of something prepared for an ovum discharged at the preceding "heat" period,—*i. e.,* several months before. Beard's theory is therefore also untenable.

It becomes necessary, therefore, to fall back on the view that the entire proœstrous or menstrual process, including not merely the growth stage but also the degeneration stage, is of the nature of a preparation for an ovum which is discharged normally (at any rate among the lower mammals) in the period immediately succeeding the proœstrum. Emrys—Roberts (1905) has recently suggested that the object of the proœstrous process may be to provide the embryo with a rich pabulum to nourish it in the very early stages of pregnancy.

Although there is still considerable difference of opinion regarding the normal time for the occurrence of ovulation in the human female, and although there can be no doubt that the two functions of ovulation and menstruation are in certain cases quite independent, there are reasons for concluding that primitively at any rate the most usual period for ovulation in women was during a definite œstrus succeeding a proœstrum, as in the lower mammals. For the period of most acute sexual feeling appears to occur gen-

erally just after the end of menstruation, while according to Raciborsky this is also the commonest time for fertile coition. (Galabin, 1904.)

There can be practically no doubt that "heat" in animals and menstruation in women is brought about through some sort of ovarian influence, while the most recent work upon the subject points very strongly to the conclusion that the nature of this influence is a chemical one and not a nervous one.

After ovariotomy it has been observed that "heat" and menstruation almost invariably cease, the small percentage of cases in which they have been recorded as continuing being probably capable of explanation on the supposition that some portion of either ovary had not been entirely removed at the time of operating. This is the more probable in view of the fact that there are numerous recorded cases in which pregnancy has occurred after removal of the ovaries, thereby showing that the removal was incomplete. (Doran, 1902.) It has also been shown that after ovariotomy in mature rabbits and other animals the uterus undergoes fibrosis or atrophy, becoming reduced to a condition such as absolutely to exclude the possibility of the occurrence of normal proœstrous changes, while in immature animals, when similarly deprived of their ovaries, the uterus instead of enlarging at puberty remains throughout life in the infantile condition. (Knauer, 1899; Halban, 1900; Marshall and Jolly, 1906.) These facts demonstrate very plainly the intimate relation which exists between the changes which take place in the ovaries and those which occur in the uterus, in so far as the latter are dependent in some way upon the former.

It was formerly supposed that this connection was through the nervous system. Thus Pflüger (1865) advanced the theory that menstruation is due to a reflex brought about by the pressure of the growing Graafian follicles upon the nerve endings in the ovary. This theory has been supported in more recent years by Strassmann (1896), who claimed to have induced a condition of "heat" in animals through the injection of gelatine into their ovaries and so causing intra-ovarian pressure. There are numerous facts, however, which are directly opposed to Pflüger's hypothesis. Thus Heape (1897) has shown that in monkeys neither ovulation nor the presence of mature follicles is necessarily associated with menstruation,

while there is a considerable amount of evidence that there is not always any correspondence between the processes in the human female.

Goetz's experiments (1874), since confirmed by Sherrington (Marshall and Jolly, 1905), show that "heat" is not caused by a cerebral reflex. In one case the spinal cord of a bitch was transected in the lumbar region. Normal proœstrum, followed by œstrus and conception, occurred subsequently, and pups were born after a full-time pregnancy. In another experiment (Goetz and Ewald, 1896) the lumbar part of the spinal cord was completely removed from a bitch which afterward came "on heat" and had pups as in the former case, thus showing that "heat" is not due to a spinal reflex. The transplantation experiments of Knauer, Halban and the present writer (working in collaboration with Mr. Jolly) show still more closely that the connection between the ovaries and uterus is not a nervous one, since in these cases the phenomena of "heat" reappeared after the grafts had acquired adequate vascular connections, while the normal nervous connections had been completely severed. It is to be noted also that the presence of a transplanted ovary in an abnormal position (*e. g.,* upon the ventral wall of the body cavity) has the effect of preserving the uterus and preventing it from undergoing atrophy or lapsing into the infantile condition.

It is evident in view of these facts that the connection between the ovaries and uterus is of a chemical character, and is therefore due in all probability to the existence of an internal secretion arising in the ovaries and exercising a definite chemical stimulus upon the tissue of the uterus and other organs, a stimulus which is most pronounced at the time of "heat," when the ovaries are in a state of greatest activity. No doubt it is possible that the ovary is excretory as well as secretory in function, but it is not easy to see how the auto-intoxication theory (which has been put forward as an alternative to the internal secretion theory in the case of certain of the other "ductless glands") can be readily applied to explain the nature of the ovarian influence.

There is a certain amount of direct evidence also that the influence is exerted by means of an internal secretion. Thus ovarian preparations are frequently administered medicinally for menopause troubles, chlorosis, etc., and beneficial results are said to accrue in

numerous cases. Further, injection of extracts obtained from animals which were in a proœstrous or œstrous state has in certain instances led to congestion of the external genital organs and other transient symptoms indicative of a condition of "heat." (Marshall and Jolly, 1905.) Similar results have also been brought about by Lane—Claypon and Starling (1906), who, however, employed ovarian extract procured from non-œstrous animals but in larger quantities.[2]

Fraenkel (1903) has put forward the theory that the specific substance which, acting on the uterus, induces "heat" and menstruation, is an internal secretion of the corpus luteum. This view is based upon nine cases in which the corpus luteum was destroyed by the cautery, in eight of which the next menstruation was said to have been missed. In our opinion Fraenkel's theory is disproved by the fact alluded to above, that in the bitch and other mammals ovulation does not occur until œstrus, and consequently that at the time of proœstrum there are no corpora lutea present in the ovaries, for the corpora lutea formed after one ovulation do not persist until the next period of "heat" (often six months after in the bitch and at a longer interval in other animals). It would appear far more probable that "heat" is brought about by an internal secretion arising in the follicular epithelial cells or in the interstitial cells of the ovarian stroma.

Fraenkel's hypothesis as to the cause of menstruation is but a part of his larger theory concerning the part played by the corpus luteum throughout the entire reproductive period of an animal's life. His general conclusions may be summarised as follows: The corpus luteum is a ductless gland which is renewed every four weeks until the menopause in the mature human female, and at different intervals in the various lower mammals. Strictly speaking there is only one corpus luteum, which is regenerated periodically in slightly different positions in the ovary. Its function is to control the nutrition of the uterus from puberty until the menopause, to prevent it from undergoing atrophy or lapsing into the infantile condition, and to prepare its mucous membrane for the maintenance of the ovum.

[2] A substance, acting as a specific chemical stimulus or excitant in the manner conceived for the ovarian secretion, has been called by Starling a hormone.

If the ovum be fertilized the corpus luteum is responsible for maintaining the raised nutrition of the uterus during pregnancy. If the ovum be unfertilized it merely produces the hyperemia of menstruation, and then undergoes degeneration until it is renewed in a fresh position.

This extended theory of the meaning and function of the corpus luteum is untenable, having regard to the fact that in many animals corpus lutea are not present for prolonged periods of the year (during the greater part of the anœstrum), and, as already pointed out, if nonexistent or in a very degenerate condition, they cannot be responsible for the hyperemia of the proœstrum. These facts, however, are in no way opposed to that part of Fraenkel's hypothesis which assigns to the corpus luteum the function of governing the fixation of the ovum, and helping to maintain its nutrition.

This part of the theory is based upon a number of experiments on rabbits in which the ovaries were removed or the corpora lutea destroyed during the first week of pregnancy,[3] when it was found that the pregnancy did not continue. These results have been subsequently confirmed in a series of experiments upon bitches and rats (Marshall and Jolly, 1905). It has been found also that if the ovaries are removed in the latter part of pregnancy, when the corpora lutea have begun to degenerate, the operation does not usually interfere with the continuance of the pregnancy. Control experiments in which the ovaries were damaged or partly destroyed in early pregnancy were also performed, and in the majority of these cases the course of pregnancy was not interfered with.

In view of the facts described above the following general conclusions have been tentatively arrived at, though it is no doubt possible that further investigation may necessitate their revision: The ovary is an organ providing an internal secretion which is elaborated by the follicular epithelial cells or by the interstitial cells of the stroma. This secretion circulating in the blood induces a series of changes which assist in causing the phenomenon of "heat" and menstruation. After ovulation, which occurs normally in the lower animals during a definite œstrus, the corpus luteum is formed, and this organ elaborates a further secretion the presence of which is essential for the changes taking place during the attachment and

[3] Pregnancy in the rabbit lasts about 30 days.

development of the embryo in the first stages of pregnancy. A point of some pharmacological importance which follows from these conclusions is that the effects produced by the administration of ovarian extract must vary according to the condition (whether œstrous, non-œstrous or pregnant) of the animals from which the extract is obtained.

REFERENCES TO LITERATURE.

Beard. The Span of Gestation and the Cause of Birth. Jena (1897).
Doran. Jour. of Obstet. and Gyn. Vol. 2 (1902).
Emrys—Roberts. Proc. Roy. Soc. B. Vol. 76 (1905).
Fraenkel Arch. f. Gynäk. Vol. 68 (1903).
Galabin. Manual of Midwifery. Sixth edit. London (1904).
Geddes & Thomson. Evolution of Sex. Revised edit. London (1901).
Goetz. Pflüger's Archiv. Vol. 9 (1874).
Goetz and Ewald. Pflüger's Archiv. Vol. 63 (1896).
Halban. S. B. Akad. Wissenschaftlichen. Vol. 110. Wien (1901).
Heape. Phil. Trans. B. Vol. 185 (1894).
Heape. Phil. Trans. B. Vol. 188 (1897).
Heape. Quart. Jour. Mic. Sci. Vol. 44 (1900).
His. Anatomie Menschlicher Embryonen (1880).
Knauer. Arch. f. Gynäk. Vol. 68 (1899).
Lane—Claypon and Starling. Proc. Roy. Soc. B. Vol. 78 (1906).
Marshall (Milnes). Vertebrate Embryology. London (1893).
Marshall (Francis). Phil. Trans. B. Vol. 196 (1903).
Marshall (Francis). Quart. Jour. Mic. Sci. Vol. 48 (1904).
Marshall (Francis) and Jolly. Phil. Trans. B. Vol. 198 (1905).
Marshall (Francis) and Jolly. Proc. Phys. Soc., Jour. of Phys. (1906).
Pflüger. Uber die Beteutung u. Ursache der Menstruation. Berlin (1865).
Sigismund. Berliner klin Wochenschr. (1871).
Strassmann. Arch. f. Gynäk. Vol. 53 (1896).

SOME PRACTICAL CONSIDERATIONS OF DYSMENORRHŒA, ESPECIALLY ITS TREATMENT

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That dysmenorrhœa is a very frequent affection with women during their menstrual lives, and that its consideration is one of more than ordinary importance to almost all physicians, must be ever recognized. It is no exaggeration to state that its occurrence is so frequent that most women are so afflicted at some time or to some degree. Such a fact becomes apparent when we recall its varieties, and the varied conditions from which it may arise.

The ordinary classification of this disease, as made by most authorities, is so clearly defined that we may reasonably conclude it is correct and thoroughly satisfactory.

A judicious expression of the pathology and the treatment of dysmenorrhœa, as we to-day understand it, cannot but be a contribution of some value. That painful menstruation is mechanical in its origin, or, in other words, that the pain is the result of some obstruction to the passage of the menstrual flow, is one of the most natural conclusions, because of its directness, simplicity, and plausibility. It also might seem the most reasonable explanation, because it is apparently substantiated by a very common method of treatment.

The practice of Mackintosh of England, of dilating the uterine canal, to remove the cause of the obstruction in this affection, reported in 1832, gave the mechanical treatment its first important impulse. This doctrine was further promulgated by the teachings and the practices of two eminent gynæcologists: Sir James Y. Simpson and Marion Sims. The latter says: "There can be no dysmenorrhœa, properly speaking, if the canal of the neck of the uterus be straight and wide enough to permit a free passage of the menstrual blood." One of the most able arguments yet offered in support of this mechanical doctrine is that of Robert Barnes of London, who concludes:

"Mechanical obstructions of the excretions are the most important factors in dysmenorrhœa. The essential condition of dysmenorrhœa is a retention of the menstrual secretion." He describes the neuralgical form of this disease with a degree of reserve and doubt.

Notwithstanding the fact that some forty years have elapsed since Simpson first introduced his method of operation, and thirty or more years since Sims advocated his, this subject has been continually agitated. No theory of painful menstruation has been so prominently brought forward; but, fortunately, it seems to me, only a few indorse it to-day.

In the light of modern gynæcology, and with the present facility for the thoroughness of a physical pelvic exploration, no one can doubt that, in a certain proportion of cases, obstructions in the uterine canal do exist, and that these may serve to create pain in menstruation. For instance, there may be abnormities of the uterine cervix, congenital and acquired. Stenoses of this canal, from the vicious topical use of caustics, especially the *argenti nitras,* while they exist, are not nearly so frequent now as heretofore; for in no manner has gynæcological practice improved more than in the local use and the choice of many medicaments to the genital tract of women.

The size of the cervical canal, and the quantity of the menstrual flux, is a matter relative and not absolute, so that much better evidence of obstruction is obtained when, immediately after the withdrawal of a small-sized sound, there rolls out pent-up secretion. It has often been claimed that the benefit obtained by dilatation with bougies, and cutting, and metallic dilators, prove conclusively the obstructive nature of this disease. The argument is faulty. Experience shows that such results are by no means uniform; and if they were, other explanations are not wanting.

The following are some of the reasons which may be advanced against any mechanical theory, and render the same irreconcilable with the clinical facts: There frequently is no well-defined relation of painful menstruation to the uterine condition. This is illustrated by cases in which there is no organic disease of the uterus, and that, too, quite frequently. Painful menstruation may be absent, or present, irrespective of the quantity or the duration of the flow; it may be present when the uterine canal is patulous, though not

so frequently as when it is diminished in size; it may be sometimes relieved by the removal of the abnormality, and again little if any changed thereby; and, in by far the greater proportion of all cases, painful menstruation is present when no obstruction can be detected. For these reasons we are forced to conclude that not only is dysmenorrhœa not obstructive in all of its kinds and in all cases, but that any impediment to the menstrual flux, as a factor in the production of pain, rarely indeed exists. The theory of dysmenorrhœa, based on mechanical obstruction, is therefore by no means demonstrated. What, then, is the nature of painful menstruation?

A certain sense of pelvic fulness and discomfort almost always attends the performance of the menstrual function; but as normal menstruation is not attended with any special pain, it is to be inferred that any catamenial period which is painful, immediately before, during, or following the flow, may properly be termed one of dysmenorrhœa. All chronic inflammatory pelvic diseases attended with more or less pain during the menstrual intervals, have some aggravation of the same at the time of the catamenial flow; but this is not dysmenorrhœa. Nor are those instances of *periodic intermenstrual pain,* cases of dysmenorrhœa; for in such experiences little or no pain is suffered at the menstrual time itself.

Dysmenorrhœa may properly be divided into the following varieties: the neuralgic, the congestive or the inflammatory, the obstructive, and the membranous. All of these forms have symptoms more or less in common; but as the types are different in their underlying morbid entities, it is necessary, for the correctness of diagnosis, as well as for the practical purposes of treatment, to differentiate each variety individually presenting itself. The dividing line between these varieties cannot always be well defined; nevertheless, they are different, and once recognized in practice, excellent practical results are obtainable.

In the first place, it must be realized that the painless performance of this monthly function in any female depends upon a good condition of the constitution at large, as well as on a healthy state of certain intrapelvic organs. Dysmenorrhœa may then be either constitutional or local in its origin and causation. The variety known as ovarian differs from the others more in its origin and location than in its kind.

In neuralgic dysmenorrhœa no special disease of the uterus or its appendages may be detected. On the most careful physical exploration no alteration in size, shape, position, consistency, or vascularity of the pelvic organs may be noticed; or, if such is observed in any case, the morbid condition is quite uncertain as to location, quality, or variety; no two cases being alike in these particulars. Again, a seeming causative local disease may be cured, but the painfulness of menstruation continues, showing that the pathological lesion, as appreciated by our senses, is not pathognomonic.

The most severe types of the malady are noticed in nulliparæ, in whom oftentimes there is no structural lesion of the uterus; possibly, this central organ of her pelvis may be somewhat undersized, imperfectly developed, or anteflexed. There is a local pelvic neuralgia or hyperæsthesia; it may be, at times, a congested condition of the uterine mucous membrane. The simple insertion of a small uterine sound, or an electrode, to and within the os internum uteri and along the mucosa above, elicits a pain identical in kind and degree with the menstrual pain itself. A slight discharge of blood sometimes follows this method of exploration, even when carefully done. This may mean that there is some congestion of the endometrium; but the pain produced cannot be the result of this condition alone, since it is out of all proportion to the degree of congestion. The sensory nerves of the endometrium must be in a state of hyperæsthesia—a local neuralgia. This hyperæsthesia is mostly at the os internum. And it is not improbable that a fissured state of the neighboring endometrium leads to a local spasm, or a contraction, such as we witness in anal fissure.

Menstrual pain thus being excited by the periodic influx of blood, it follows that the greater the tension and the rigidity of these tissues, the more the pain—an explanation not improbable, because of the greater relative frequency of this form of the disease in the unmarried and the nulliparous. A similar unyielding character of tissue is present in some cases of chronic metritis. Undue vascular tension and compression of end nerves always excite irritation. When the flow is well established, the local swelling subsides, and this tension is relieved.

It is reasonable to attribute the painful menstruation accom-

panying versions and flexions, to the chronic congestion, which as often antedates as results from the displacement. In flexions of the uterus, this organ, turgid from an obstructed circulation, its nervous supply in a state of irritability, will resist the expansion of the physiological menstrual hyperæmia more than a normal structure; and hence there is pain. So potent are these two factors in pathogenesis that, in any given case of seeming or real obstruction within the uterine canal, it is difficult to estimate how much may be attributed to the latter. That obstruction is not altogether concerned seems demonstrated by the fact that those means which are most useful in diminishing local blood supply and tenderness—lack of attention to the displacement *per se,* and no change in the position following—are oftentimes efficacious in relieving pain.

Dysmenorrhœa is, then, a functional disorder of the uterus, and, in its essential and underlying nature, a neurosis. It presents many features analogous to other visceral neuralgias. The temperament of the dysmenorrhœic is highly sensitive and impressionable. As pain is a matter not absolute, but relative to each individual, constitution, or state, there is in this disease a special susceptibility to pain, extensive in character, and at times seemingly subjective in its nature. The local neurosis is generally an expression of an impaired, disordered, or highly nervous organization.

Various blood states conduce to this feature of the affection. A want of general development of the body at large predisposes to this neurotic habit; so does an imperfectly developed sexual apparatus. It is a rational inference to regard the painful menstruation so often accompanying malformations of the cervix and congenital flexions, as in some part dependent upon corresponding defects in the physical organization of the corpus uteri and internal genitalia, although the measurement of the uterus made with a sound, may not be below the normal standard. These are cases of primary dysmenorrhœa, antedating all structural lesions of the uterus. They embrace a large class in the unmarried, no small percentage of which, unless early relieved, become permanently sterile before a certain amount of organic change has occurred in this organ.

In no variety of dysmenorrhœa, more than the neuralgic, is it so important to estimate the standard of the patient's general health. There is present a local neurotic state of the pelvic organs, provoked

to the excitation of pain by the stimulus of the physiological pelvic congestion incident to the oncoming menstruation. The pain is usually increased by the presence of the hæmorrhagic flow within the uterine cavity. The local neurosis, an expression of the nervous system in which there is an exalted sensibility, shows itself by some general hysterical phenomena; a spinal irritation, a neurasthenia, and a local or general neuralgia. Pain, like age, is relative. The cause may be the same, but no two patients suffer alike. Anæmic and chlorotic states of the blood must always predispose to neurotic dysmenorrhœa.

Rheumatism and gout are direct exciting causes. The rheumatic form of the disease much resembles the purely neurotic. All habits of the body conducive to indolence, as a want of physical exercise of faulty ways of dress, indirectly lead, by enervating the nervous system, to dysmenorrhœa. Hence the disease is relatively common in the upper classes of society. General ill-health retards an easy and physiological disintegration of the menstrual decidua. Truly, may it be affirmed that this affection is a penalty of a poor inheritance, a defective hygiene, a forced education, and the false stimulus of our modern and artificial life.

The pain experienced may vary in every possible manner, as to time, duration, severity, and location. Alternate periods are at times less painful. Some cases are so pronounced that suffering is felt at the very inception of this function at puberty, continuing with increasing force for years, until it becomes most severe. Such periods are always most dreaded. They incapacitate one for at least a week each month, and are followed by much prostration. Neuralgic dysmenorrhœa then becomes a potent force to undermine a girl's health, and thus makes her an easy victim for chronic invalidism, hysteria, and neurasthenia.

Pain in dysmenorrhœa is not invariably located within the pelvic regions, especially in neurotic instances, for it is at times felt in other parts of the body. Usually it comes on soon after the commencement of the flow, is most severe during the first day, and diminishes during the second day and the last. The discharge may be scanty or profuse, or consist of clots. The severity of the pain seems, for the most part, to be in an inverse proportion to the quantity of the flow. In the congestive or inflammatory type of the dis-

ease, the sufferer feels more at ease after the flow is well established, a clinical fact contrary to what is often experienced in the neurotic variety.

There is a want of conformity between the seeming causative lesion or abnormality, and the symptoms which are manifest. Not only, as stated, may there be dysmenorrhœa when no abnormal conditions of the uterus, as to size, shape, position, or condition, can be detected; but, as previously stated, well-defined abnormalities of the uterus, as the pin-hole os, the elongated and conoid cervix, the contracted cervical canal, the flattened and ill-developed uterine body, and flexions, may be present without dysmenorrhœa. Stenosis of the os externum, marked in kind and in degree, resulting from chronic inflammation, or the injudicious use of certain caustics, and well-defined acquired flexion, are not always attended by menstrual pains. Instances, on the other hand, are not wanting of uteri with patulous canals associated with much dysmenorrhœa. Clinical evidence points to the conclusion that the neurotic feature is the chief, and only cause in many cases, and that the neurotic element is manifested to a greater or less extent in all, irrespective of the variety of this disease.

The pathology of the so-called membranous dysmenorrhœa is a matter of some dispute. It seems most reasonable to accept the view advanced by Raciborski and Simpson; viz., that it is the desquamation, or the exfoliation of the intracorporeal uterine mucous membrane—an exfoliative endometritis. Klob, whose opinions have been widely accepted, says that the false membrane, expelled in shreds or in complete sections, is an exudation from an old chronic endometritis. Braun accepts this view. A microscopical examination of the membrane may be needed to differentiate the material from blood clots, polyps, diphtheritic exudations, or products of an early conception. It is necessary to make a diagnosis first, and then to institute an active local or general treatment of a proper kind, to effect a cure of this most stubborn malady.

The prognosis of dysmenorrhœa is for the most part very favorable. The longer the duration of the disease, however, other things being equal, the more difficult it is to effect a cure. The difference in the curability depends largely on the fact that the impressionability of patients to pain becomes more and more marked by time.

Nothing increases the susceptibility of the nervous system to pain so much as the constant use of anodynes, particularly opiates in some form, by many of these patients.

The neuralgic variety of the affection is more amenable to treatment now than formerly. The great majority of cases, irrespective of kind, degree, or duration, are entirely curable. Again, it may be said that the congestive or the inflammatory type is easily relieved; the obstructive is controllable, but the membranous is the most obstinate to combat.

The successful management of this disease depends upon the proper recognition and the removal of the cause. Based, as it should be, on the conception of the nature of the pathology of individual cases, it is of paramount importance that this be sound and correct. In all cases of painful menstruation, in young unmarried women, the treatment should be purely constitutional, at least for a time, until the necessity for a local examination is clearly and justifiably apparent. Fortunately this urgency rarely arises. At this age of her life and in this social condition, except for congenital abnormality, there is seldom any local disease, or, if one exists it is too trifling and too insignificant to require local treatment.

We may first consider the treatment required for the periodic pain, and then the management of the menstrual intervals. For the attacks of pain no local treatment is needed, except what the patient herself or her nurse may employ. A great many remedies have been prescribed to mitigate the menstrual pain, but reference is here made only to such as I have found very useful, and never injurious.

In the first place, permit me to condemn, in the most unqualified terms, the use of any opiates, and likewise the recommendation of any of the so-called tar products. Any one of these drugs may assuage the suffering for the time being, but in no sense will they prevent its return; rather will their further use be necessitated by the force of habit. Almost all women who have become habituated to the use of opiates and other narcotics, have been enticed unknowingly to depend upon them, by having these medicinal agents prescribed for them too frequently or too continuously. It is the easiest matter in medical practice to fall a victim to their use—always baneful, ever difficult to surmount—a result which is worse than the original mischief for which they were prescribed.

The tincture of pulsatilla is an excellent remedy in not a few cases. An increasing experience with it gives me greater faith in its virtues in the neurotic types of the disease in young women, although it is not contraindicated in any form. It is best given in 10-drop doses, three times a day, for at least three days preceding the expected inception of any painful period; and then it should be continued in similar doses, but given more frequently (every one, two, or three hours) at the menstrual times, if pain is then present. The tincture of *Actæa cimicifuga racemosa* may be prescribed in a similar manner. It, too, is clearly indicated for the neuralgic form of the disease, and probably is beneficial in cases of apparent rheumatic origin. It is a very efficacious remedy, quite useful when it is given steadily three times a day during the whole interval, and at spaces of time of one to three hours during the menstrual period, if there is pain. As a matter of course, the salicylates are most serviceable in cases distinctly rheumatic, but they tend, when given during or near the menstrual time, to make the uterine flow freer and less painful. Guaiacum, in the ammoniated tincture, is useful in the neuralgic and rheumatic forms. It is given just before and during the periods, and may be administered in milk or small quantities of whiskey at short intervals. Fenner's tincture—gum guaiacum with the mercuric bichloride—was a remedy in very common use some years ago. All of these remedies act best when administered in small, but repeated doses.

A combination of cimicifugin, helonin, and caulophyllin, in pill form, three times a day, during the intervals and menstrual times, has been found very helpful when the dysmenorrhœa is associated with a delayed and scanty flow. The bromides of sodium and ammonium are indicated in the ovarian types of dysmenorrhœa. The bromides tend to diminish in quantity the menstrual flux. Cannabis Indica is a nerve stimulant, an anodyne, and an antispasmodic. It acts in a measure like ergot, but more promptly and energetically, if the drug is pure. It would seem to be indicated in cases where the ovarian neuralgic condition is present. It is certainly to be chosen in preference to opium.

There are cases accompanied with a vasomotor spasm, characterized by a paleness and a coldness of the surface of the body. Such cases present indications for the use of glonoin or nitro-

glycerin; to be administered in 1-drop doses, well diluted, every few hours. By its use, the periphery of the body becomes warmer and of a better color, and pain is relieved by an equalization of the circulation.

We come next to the consideration of the proper treatment during the menstrual intervals. All cases must be judiciously attended to each month. This treatment has reference to the removal of the causes, and then to the rectification of the underlying conditions, local or systemic, intrapelvic or constitutional. Herein lies the most rational consideration of the whole subject. Any constitutional treatment implies, first, the correction of all defective hygienic habits. Every dysmenorrhœic patient should be cautious in the selection of diet, and in the correction of any error of the digestive functions. She needs to eat well, at proper intervals, to improve her general health, and to fortify herself against the inroads of pain. A strong and healthy body feels any local pain less, and experiences but little depreciation by its return. The patient's garments ought to be loose, comfortable, and so adjusted as not to impede sufficient bodily exercise in the open air. Daily baths, for the most part in cool water, not only secure cleanliness, but free the cutaneous action and invigorate the whole body. I am inclined to prescribe for anæmic, debilitated women a daily bath in cool water, medicated with a good proportion of sea-salt, and followed by vigorous friction. The patients not only feel much better, in that the circulation of their blood is improved, but they eat better, and their nutritional processes are signally made more active. While a reasonable quantity of physical exercise of some kind, about the home or in the open air, is daily to be practised, some mental occupation and exertion are not to be neglected. The bowels need to be evacuated freely each day. Systematic cholagogues and saline purgations are called for, in the congestive and inflammatory varieties of dysmenorrhœa.

Marriage is by no means contraindicated. Many women, it is true, are sterile because of their dysmenorrhœa; but if fertility should follow marriage, then pregnancy, parturition, and lactation may cure the disease. Every dysmenorrhœal patient who is married and who for a time is sterile, should be examined in order to ascertain the exact condition of her intrapelvic organs, and, if necessary, be treated locally in an appropriate manner.

Many, if not most, patients suffering from dysmenorrhœa are more or less anæmic, and in consequence a scanty menstrual flow with them is more common than a profuse one. Hence, iron in some form is a very good preparation to begin with, after the disordered digestive functions have been regulated and a good diet commenced. The best preparations of iron are the dried sulphate, the carbonate, and the muriated tincture. They are to be taken only during the menstrual intervals. The system may be further strengthened by other tonics, such as the bisulphate of quinine, strychnia, the phosphates, and cod-liver oil, given alone, or in combination with the iron salts. Arsenic is the choice remedy as a tonic, if the menstrual flow is profuse; and the mercuric bichloride, in minute doses, dissolved in the fluid hydrastis, if there is any chronic endometritis.

All excitements, both general and local, as well as any undue sexual intercourse, dancing, and the use of the sewing-machine should be abstained from.

Morbid alterations of the uterus and the periuterine tissues and organs are to be remedied, as far as possible, during the menstrual intervals, by proper medical and surgical treatment. One of the most common of these morbid conditions is chronic endometritis. The cervical dilatation, followed by thorough intra-uterine curettage with the sharp instrument, removes morbid structural changes of the mucous membrane more promptly and more thoroughly than any intra-uterine medication. Aseptically done, with reasonable amount of rest afterwards, it is an operation at once safe and almost always beneficial. It may need to be repeated; it certainly will, if the painful menstruation is of the membranous kind. Possibly, the benefits from dilatation of the cervical canal, in some of these forms of dysmenorrhœa, may be attributable to thorough stretching of the circular uterine fibres about the os internum, as dilatation of the rectal sphincter relieves fissures of the anus.

The question arises at times, as to whether öophorectomy is ever justifiable to cure dysmenorrhœa. Removal of the ovaries by abdominal section will almost always stop the return of the menstrual periods. In so doing it will cure future menstrual pains. But is this operation ever, under any of these circumstances, to be recommended? In reply, we must speak in the negative. Never should

normal ovaries be extirpated simply to stop menstruation and prevent recurrences of dysmenorrhœa. Fortunately, I have never been forced to this position. I have felt justified in performing salpingo-öophorectomy in cases in which there was dysmenorrhœa, but the operations were for the removal of the diseased tubes and the ovaries, and not for the cure of the painful menstruation alone.

Let me now mention what seem to be the appropriate measures for the treatment of the remaining forms of this painful affection. What can we do for the chronic inflammatory lesions which are without any doubt the chief underlying bases of trouble? Usually the inflammation is in the endometrium, and these inflammatory lesions are chiefly confined to the body of the uterus. The cervical canal is not concerned in the disorders of menstruation. Intra-uterine medication, by ingestion or by injection, is painful, tedious, and not altogether safe; generally it is quite unsatisfactory in its results, and as a single remedy it is now almost abandoned. Preceded by a thorough dilatation and a sharp curettage under anæsthesia, if done under strict asepsis, it is safer and less painful, saves time, and is in every way to be preferred to the older method. Some intra-uterine medication may be given at the same time, or soon after. The best results follow this minor surgical procedure. Not only are the diseased conditions of the whole endometrium improved or even cured, but these beneficial results extend to the uterine parenchyma, and affect favorably the tubes and ovaries, so often secondarily involved in septic or specific infections. After an experience of many years with intra-uterine medication, my choice of local medicaments is Churchill's tincture of iodine, the iodized phenol, and ichthyol. The use of these drugs should only follow dilatation, with the expanding dilator and the free scraping of the endometrium with the sharp curette. Finally, the whole uterine cavity should be packed. Not sooner than one week after this minor operation one or the other of these topical agents may be utilized locally.

Now as to cervical obstructions. Believing that stenosis of the uterine canal exists much oftener at the external than the internal os uteri, it can readily be understood why sterility is far more frequent and persistent than dysmenorrhœa. It is easier for the menstrual discharge to escape from the uterine cavity than for the

spermatic fluid to gain entrance to it. We must open up the stenosed, or strengthen the flexed, canal. It is well-nigh impossible for sterility to continue with any woman, in a married life of a period over five years, without its causing local disease of this central pelvic sexual organ; viz., a catarrh, parenchymatous congestion, displacement, and sympathetic disorders. So vascular are these organs that they cannot be subjected indefinitely to the hurtful influences of oft-repeated influxes of blood, without suffering some morbid disturbances of circulation. Just here again, free dilatation, sharp curettage, and thorough packing, with possibly, some intra-uterine medication, promise much, not only for the dysmenorrhœa, but also for its associated sterility. Congenital conoid cervices, with a pin-holed os externum, are benefited by these procedures.

But we should ever bear in mind that the os and the lower cervical canal will at times return to their former faulty size and shape, before impregnation ensues. A cervical canal permanently patulous is best obtained by intra-uterine galvanization, with the cathode electrode placed within the uterus. Theoretically, electricity appears to be clearly indicated in many cases of painful menstruation; experience has fully substantiated this view. Commencing with the simplest of all methods and means for the use of electricity, let the intrapelvic electrode be placed in the vaginal canal, in its posterior cul de sac, against the uterus. By choice, let this be the positive pole of the primary Faradic coil, or either pole of the secondary coil. The latter is often preferable, because of its recognized analgesic powers; the longer and the finer this coil of wire of the generating current, the better. Without doubt there are cases of this disease, which are relieved simply by the external method of application of both poles. How much of this is psychical, it is difficult to estimate, but a long continued and severe dysmenorrhœa which has resisted constitutional and a well selected medicinal treatment, calls for electricity, and if this fails the method of intra-uterine galvanization should be utilized.

The following has long been my method: By virtue of the anæsthetic effects of the positive pole of the galvanic current, it should, in these pronounced and intractable cases, be applied within to the fundus of the uterus; while the negative electrode, of good

size, is adjusted over the lower abdominal surface. The intra-uterine electrode is constructed of flexible copper wire, silver plated, the size of a sound; the negative electrode is made of copper plate, covered with a double layer of soft flannel. One, not more than two, seances each week, during the intermenstrual intervals, with a strength of current from 10–25 milliamperes, for from ten to fifteen minutes each, are all that is sufficient. It is desirable not to utilize very strong currents, or to apply them too frequently or too long. Each treatment should always be done with the most aseptic precautions. It is well to have the patient maintain, for a half hour or more afterwards, the recumbent posture; or, the whole treatment is done in some hospital, where she is at rest. All danger is thus minimized by care and skill. If the os externum and cervical canal are larger than normal, another special indication is had for the internal pole to be the anode; but should the uterine canal be congenitally or artificially contracted in its calibre, the cathode is the choice. The good results following one to two months' treatment, one application a week, in virgins and in sterile married women, are the highest awards we can offer for this valuable method.

The membranous variety of dysmenorrhœa may be managed in the same way. Favorable results have been reported from repeated sharp curettements of the uterus, with certain internal medication. But, after the trials made by repeated sharp curettements, permit me to suggest a faithful use of intra-uterine galvanization. This form of electricity is an alternative to curettage for membranous dysmenorrhœa, and will cure when the latter has failed. The intra-uterine pole should be the cathode.

There is no class of cases which the physician is called upon to deal with, that requires more patience in the treatment and more skill in the diagnosis than those in which pain preceding, during, and subsequent to the menstrual flow, constitutes the symptoms. Electricity alone can do much to relieve and even to cure. Where electricity has been used in combination with suitable local and general treatment, brilliant results have been obtained.

Pediatrics

GONORRHŒA AND SYPHILIS IN INFANCY AND EARLY CHILDHOOD *

BY THOMAS MORGAN ROTCH, M.D.

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I HAVE been requested by the Executive Committee of your society to address you on "venereal disease among children," and I thank you for giving me an opportunity of expressing my views on this important subject. As the time of life with which I propose to deal is that of infancy and early childhood, the name "venereal" would be inappropriate and a misnomer as applied to this class of cases. Venereal means "arising from or connected with sexual intercourse." This is practically unknown in early life. "Venereal" is a collective term including chancroid, gonorrhœa, and syphilis. I shall speak only of the latter two diseases, as the former has but little comparative significance in our present discussion. I shall, therefore, if you will allow me, adopt as the subject of my address, "Gonorrhœa and Syphilis in Infancy and Early Childhood."

There are many questions, both moral and physical, connected with the subject of gonorrhœa and syphilis in early life which may well and ought to come within the province of the physician. Physicians are brought into the closest connection with families where spectres of what may exist, are feared to exist, or may exist in the future, continually cast shadows of distrust and unhappiness through generations. In each of these questions there are also many important sides to be discussed—all important, but far too many to be dealt with lightly in one evening. I shall, therefore, confine myself to a few words emphasizing the self-evident

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proposition that it is the dissemination of a precise knowledge of gonorrhœa and syphilis which will best enable us to combat the ravages of these diseases, and stamp them out.

Knowledge is indeed here a necessary agent and a wonderfully efficient coadjutor in a contest with these diseases. True pictures of these diseases should be taught, shown to, and thus recognized by the laity as well as by physicians. Definite knowledge of their manifestations and vivid portrayals of their direful results, purposely made hideous, should be brought before the people; for the possibility of causing, in innocent individuals, syphiliphobia is too unusual and too remote to warrant us in staying our hand for the benefit of the few to the detriment of the many. It is needless to tell this audience that these diseases are very prevalent: statistics are not needed to prove this statement in a subject where too much time cannot be spent in discussing the prophylaxis and treatment of the many cases which are continually coming under the observation of physicians.

Even if statistics did not show a large preponderance of these diseases, their power to spread rapidly would make their suppression of vital interest. Is it, however, known how prevalent is gonorrhœa in infants, and how numerous are the cases of hereditary syphilis which are brought to our children's clinics to be treated for eczema or other local diseases of the skin? I think not—and it would be well if all physicians who are working, mostly among adults, should appreciate this fact, and spread the knowledge of this state of affairs among the laity, to whom it may never have occurred that young babies are a source of infection.

Just as it is important that a crusade should be carried out against that far-reaching and deadly foe of the human race—the bacillus of tubercle, so should we endeavor to stamp out in our communities the organisms which produce gonorrhœa and syphilis. The question is, how shall we in the best and wisest way make our attack? From a physician's point of view, I should say that our mission is to understand first thoroughly all that is known about these diseases, and that not only physicians should so understand, but that the public at large should be informed of the nature of the diseases, of their insidious spread, and of their unfortunate results. No cloak should be thrown over the history, course, and results of

these diseases. Pus should not be allowed to burrow, but should be kept on the surface. Half the battle has been won for morality when the evils which are undermining it are brought to the surface and exposed to the searchlight of the public eye, educated and assisted by plain scientific truths.

For physicians it is a positive duty to understand thoroughly and study at least the diagnosis of these diseases and their far-reaching effects; since no matter in what branch of medicine or surgery they are working, these diseases often play a great rôle. They make an otherwise plain case obscure, and insidiously undermine the tissues to such an extent that the usual and appropriate treatment for the especial disease—whether it be the bones, the eye, the ear, the brain, the lung or heart, the liver, or other abdominal organs—proves to be totally inadequate. Perhaps, also, when too late to control tissue destruction, someone who understands the true cause which is baffling a brother physician's usually successful treatment, tells him how, if he had recognized the etiology of the case, he could have saved it. This, I assure you, is continually occurring. Is it asking too much, that every physician, in the interest of science and humanity, should acquire a knowledge of these most Protean of diseases, sufficient at least to make him suspicious of their presence in doubtful cases. I truly believe that this is something which cannot be impressed too strongly on physicians.

I shall not, of course, in what I have to say about these diseases, give any details in the sense of teaching, as that would be unnecessary and out of place before such an audience as is present here tonight. What, however, I have thought might be of benefit and interest for the uses of your society, is a short practical résumé of my experience in these diseases, and the views which, deduced from this experience, I hold concerning their public management. I may say that my opportunity for observing these diseases is a fairly large one, and affords most advantageous conditions for their study. Having an unusually close control of the medical service of two hospitals —one devoted entirely to infants and the other to children—and with a material amounting to some 10,000 in the year, a number which if carefully and systematically studied is ample for general conclusions, I ought as a public citizen, as well as a physician especially interested in and working among children, to have deduced

from this experience, which includes an opportunity of twenty-five years, at least some rational ideas in connection with their extinction. So important have I held the study, especially of hereditary syphilis, that from the very beginning of my teaching at Harvard, over twenty years ago, I have made a special effort to make this teaching prominent among the other diseases of early life. I believe that it is the duty of the teachers in all our great medical centres to do likewise, and thus to impress upon the physicians of the future their responsibility in regard to a feature of medical study which should, on broad general principles, working for the good of the state, rank second to no other disease, not excepting tuberculosis. The bacillus of tubercle rarely weakens a brain which may be of great use to the state, at least leaves a human being and a good citizen respectable and usually inspires a strong desire to live. The organism of syphilis, on the contrary, even if not weakening the brain organically, decreases its proper use by constant fear of what next dread symptom may arise and increase the horror of being stamped as a bad citizen, as possibly a loathsome creature to look upon, avoided by his fellow men and women, and damned even worse than the leper, who at least may be respectable. The organism of syphilis, and I say this, knowing and appreciating the great results which have been obtained in certain cases from treatment—treatment, however, which is often of no avail in the many broad questions which arise outside of the immediate welfare of the individual—I repeat, the organism of syphilis not only does not inspire a desire for life, but it induces the desire for death, and in many cases its unfortunate victim would gladly seek for the entrance, and rush into that Inferno of Dante, mocking at the inscription above the door: "All hope abandon—ye who enter here!"

Again, in regard to experience in and the teaching of hereditary syphilis, I have always believed that it is not the specialist in syphilis who sees most of or can judge best in dealing with infantile syphilis, though, of course, his advice is most valuable as a consultant. The responsibility is thrown over and over again on the physician who sees these infants in their homes, sufferers from the many ailments of infantile life.

It is under these circumstances that a physician can best really know all that ought to be known in regard to these cases, fully

understand them, and properly refer them for treatment. So firmly do I believe that the teaching of infantile gonorrhœa and syphilis should be enunciated in a medical curriculum, that I pay just as much attention to it in my Harvard lectures and in my clinics as I do to scarlet fever and measles. More than this, I have always unhesitatingly had cases of hereditary syphilis admitted to the wards of both hospitals, which, of course, we cannot do in cases of the other infectious diseases, such as diphtheria, scarlet fever, measles, chicken-pox, and whooping-cough. Although opposition at times arises from the extremely contagious nature of the disease, yet this is in itself useful, for nurses and students are thus taught to understand the nature of the contagion, and that it is not disseminated through a ward as are measles and scarlet fever, but that danger from it can be entirely eliminated if proper precautions are taken. This is also a very important matter in connection with the instruction of the trained nurse.

I have never known gonorrhœa or syphilis to be communicated by a syphilitic infant or child to either patient or nurse in the open wards of the Infant's or Children's Hospital, where they have been treated during a service of twenty years.

Gonorrhœa and syphilis belong to a class of contagious diseases where the infection, though it may be mild, is, as a rule, intense and often virulent.

GONORRHŒA

In regard to what physicians and, in fact, the laity should know regarding gonorrhœa in early life, it is well, perhaps, to enumerate certain points in connection with this disease. Although it is just as infectious as in later life, and although its effects may be quite as disastrous, it naturally does not assume the same position as in later life.

It should be known that the infection is produced by a specific organism, the gonococcus of Neisser. This organism is easily recognized by smears taken from the infected membrane, stained with Gram's method, and the growth then differentiated by its shape under the microscope. The organism will not grow in ordinary media, and therefore either the fluid of a hydrocele or human blood agar has to be used. Most animals are immune to the gonococcus of Neisser. The most common nidus for infection and transmission

are secretions from the infected surfaces of the vagina, urethra, and eye. It is, however, not only these localities which can be affected, as it has also been found in certain individuals to infect any mucous membrane, such as the mouth, where there has been a previous abrasion. Blindness has resulted from infection of the eyes, and a gonorrhœal urethritis has been found in babies as young as eighteen months, and also a resulting urethral stricture in a boy five years old. The most common locality in female infants is the vagina, and the form a vaginitis.

The organism having once gained entrance to a hospital, may spread rapidly and become epidemic. Sometimes as many as 10 per cent. of the patients in an infants' hospital have shown the infection, which usually starts with the admission of an acute case. This, of course, points to the importance of a routine examination being made of all vaginal discharges. From 80 per cent. to 90 per cent. of the mothers of these infected babies were found to have a leucorrhœal discharge, and the nursing baby has been known to have gonorrhœa of the mouth from infection through its mother. It is easily seen how such a source of infection as this might cause untold trouble; for the nurse or other members of the family can be infected by a baby that was supposed to have simply a slight irritation about the vagina.

Although as a rule there are no secondary lesions following this class of cases, yet it must be understood that serious complications may arise. As a result we have endocarditis, pericarditis, and peritonitis, or the patient may be attacked with an arthritis, usually of the multiple form, and sometimes with the constitutional symptoms of pyæmia.

The infection is carried directly by the hands, or can be obtained from articles of clothing, towels, bed linen, napkins, thermometers, tubs, sponges, water, and anything else of the kind. The gonococcus is often extremely difficult to get rid of, as is shown by the way in which it lasts in hospitals, causing epidemics in hospitals or wherever young children are gathered together. The most prolific source, however, and one which is the most difficult to obviate and eradicate, especially in hospitals, is when, as is often the case, the disease is spread in infantile cases by means of the napkins. In these cases it has at times proved impossible to get rid of the infection until un-

usual care was taken in the washing and disinfecting of the napkins.

It is now well known that many cases of vaginitis, formerly supposed to have been caused by animal parasites, such as the ascaris vermicularis, have been found, on investigation, to be an infection produced by the gonococcus. We, therefore, do not consider that a correct differential diagnosis of vaginitis has been made until the vaginal discharge has been carefully examined for the presence of the gonococcus. The insufficient knowledge that this means of infection exists, will cause its source to be overlooked; and entirely innocent people, as has often happened, have been stamped with the stigma of gonorrhœa; for the infection can be conveyed by the hand as well as by any other part.

This disease, then, is far more widely spread than is usually recognized, not only by the public, but by physicians; for it is hard to make moral and respectable people believe that a little innocent baby, a few months old, is the means of producing gonorrhœa in a number of adults. These facts must be known, however, before we can successfully stop the spread of a disease, which, it is hardly necessary for me to tell this audience, may produce not only blindness but sterility in both sexes.

It seems to me that this society, from a humanitarian stand-point, should pay attention especially to this fact. Perfectly innocent people may be found to have the disease, through no fault of their own, excepting ignorance. They have not contracted the disease necessarily from another adult, but may have been infected at a time when they were caring for, and protecting from harm, that most helpless period of life—babyhood. Certainly a knowledge, and an open knowledge, of these diseases is absolutely necessary for the protection of the innocent, as well as for the punishment of the guilty.

SYPHILIS

Syphilis has been studied in its primary and secondary lesions, and has been recognized as infectious ever since the sixteenth century.

In 1881 Aufrecht announced the discovery of a special variety of cocci in syphilitic condylomata. In 1885 Lustgarten announced the discovery of the bacillus of syphilis, but this is now known to be only the smegma bacillus. Since then, a large number of germs

and micro-organisms have been described, and on March 3, 1905, Schaudinn, in Berlin, discovered a spiral organism in a secondary papule of a case of syphilis and named it "Spirochæta pallida." Since then a large number of investigators have studied the organism and the literature of the subject is quite extensive. This organism has been found in congenital syphilis and in the blood of cases of secondary syphilis. It seems to be constantly present in primary and secondary lesions. The organism is found in the tonsils and inguinal glands and in condylomata.

Dr. Flexner is carrying on a series of investigations in the Rockefeller Institute. His opinion is that while we are not justified in declaring that the organism is pathogenic for syphilis, the investigations certainly point towards the fact that we are on the right track, although we should still be conservative as to our opinion about this organism.

Although all forms of syphilis are produced by the same infecting organism, we, for the purpose of clearness in our clinical teaching, separate the subject into two divisions: (1) the acquired and (2) the hereditary, or, as it is sometimes called, the congenital. In the acquired form the organism is transmitted by actual contact, directly from one individual to another; and, according to the point of contact, produces what is called the primary lesion. Following this primary lesion arise later certain well recognized secondary lesions, again to be followed by lesions which, after many years, may appear and are called tertiary. This acquired syphilis in all its forms—primary, secondary, or tertiary—may occur at any age, whether it be in adult life or in infancy. It is, however, excepting in the slums of great cities, rare in comparison with the second division spoken of above; and it is the hereditary form which is of interest to us to-night and which we are especially discussing.

This form is transmitted to the offspring through its progenitors, and in this sense the syphilis originates in intra-uterine life. That is, the primary infection is of the progenitor, and the offspring, therefore, does not show the primary lesion, but presents the same lesions as the secondary or tertiary manifestations of the acquired form. At times the secondary symptoms are so slight as not to be noted, and the infant becomes a child before it is recognized to have syphilis. It is these cases which are spoken of as retarded syphilis.

The organism of syphilis is the most Protean of any known disease in its clinical manifestations, in the variety of its lesions, in the degree of its intensity, in its destruction of tissue, and in its far-reaching results. The symptoms may often be very obscure, but again may be represented by definite lesions of any of the organs. According as the syphilis is present in its secondary or tertiary form, the especial organ or tissue presents one or another type of disease: thus, when the eye is affected it is an iritis which is most apt to occur in infancy, and interstitial keratitis in the later tertiary form. Again, in infancy, when the bones are affected, it is especially an osteochondritis which is present, while in childhood the pathological condition of periostitis is most common. The mental development is retarded, the growth in height interfered with, and the unfortunate condition of infantilism is liable to occur. As a result of hereditary syphilis a peculiar form of deafness, intractable to treatment, may occur in childhood. Many deformities, such as those of the nose, and sequelæ, such as blindness, may be terminal results.

The most frequent, and perhaps in relation to our discussion to-night the most important, are the various lesions of the bones. The most common lesion of bone disease in hereditary syphilis is an acute epiphysitis and its accompanying periostitis, leading later to separation of the epiphysis and to a pseudoparalysis, simulating also in many cases fracture. To those who are not especially acquainted with the many ways in which syphilis simulates other diseases, these lesions of the bones are peculiarly apt to be misleading. I myself have seen instances where separation of the epiphysis of the wrist in a young infant has been mistaken and treated for fracture, the thickened periosteal exostosis being also mistaken for a callus. The lesions, however, which are most likely to be confounded with syphilis of the bones are the tuberculous. It is, of course, exceedingly important to differentiate between syphilis and tuberculosis, as many interests of a most opposite character are involved, and many questions of the future life of the patient, which differ radically according as one or the other organism is to be dealt with or to be considered. It is in these cases, which are often obscure, that especial means for a differential diagnosis should be employed. It may, therefore, be worth while to speak of a few of the differences in the living pathology of the bones which can often

only be differentiated by the use of the "Röntgen ray." There is no doubt that not only every means in our power should be employed to recognize obscure cases of syphilis on account of their treatment, but also because it is important to relieve, if possible, the patient from the stigma of syphilis by proving that some other organism is producing the obscure symptoms.

It should be recognized that a radiograph, in order to give precise and reliable information, should be taken not only by a mechanical expert, but by one who has made a study of the bones at different ages, and knows at what periods during infancy and childhood the cartilage becomes bone: as, for instance, when the epiphyses become ossified. Knowing the normal conditions, which, indeed, differ greatly at different ages, we can then say in a given infant or child whether it is a normal or abnormal condition presented by the ray; and then, again, whether we are dealing with a fracture, osteomyelitis, scorbutus, tuberculosis, or syphilis, for all these diseases in their bony changes at times simulate each other closely.

It would be out of place to speak to-night about the finer changes in the bones in the different diseases, as I am merely pointing out, as a suggestion for the line in which our differential diagnosis should work in the future, certain salient and macroscopic pictures which can be detected by the "X-ray" during life.

The shadow at once shows whether a fracture, a separation, or a dislocation is present. It also shows the destructive process of an osteomyelitis, starting, as a rule, first with infection of the periosteum, going on to a gradual distention of the periosteum followed by destruction of both periosteum and cortex. Secondly, the process begins in the medulla with infiltration followed by absorption of the lime salts and a resulting sequestrum formation. It shows the superiosteal hæmorrhage of scorbutus, as distinguished from an exostosis of the bone, or a tumor. It distinguishes between the destruction of bone cortex found in tuberculosis, and the marked thickening of periosteum, layer on layer, significant of syphilis.

I have a few radiographic pictures to show, which illustrate my idea as to how we should equip ourselves in dealing with the many-sided question of syphilis, where exact knowledge is a prerequisite.

These radiographs were taken by Dr. A. W. George, whose

unusually excellent work as Röntgenologist at the Children's Hospital has enabled me to make a great many valuable observations by means of the X-ray.

Fig. 1 shows the leg of a boy eight years old. There was no family history to aid in the diagnosis or pronounced symptoms of syphilis, but the child was evidently suffering from some obscure disease. The shadow showed the extreme thickening of the periosteum characteristic of a retarded syphilis.

Fig. 2 shows the arm of a child five years old who was markedly atrophic, and with a history of syphilis in the mother. The ulna shows the marked periostitis without apparent destruction of cortical bone distinctive of syphilis. At the wrist was a swelling distinctive of a secondary osteomyelitis produced by the staphylococcus.

Fig. 3 shows destruction of the cortical bone and a slight cavity in the fourth metatarsal phalanx of the foot in a girl twelve years old. Suggestive of a tubercular process, or possibly a benign bone cyst.

Fig. 4. This represents the hand of a boy nine years old. It shows a tubercular dactylitis of the first phalanx of the middle finger.

I have already referred to the stigma which results from the brand of syphilis where syphilis does not exist. This, I assure you, is not uncommon. In the varied and extensive interchange of opinions and diagnoses which have come to my lot during the last twenty years, I have been much struck by the fact that the laity believed frequently that such disease as acne rosacea, and tuberculosis of the skin, with the various lesions formerly known as lupus, indicated syphilis, and that individuals with these diseases are looked upon as degraded. This not only happens with the laity, but I have again and again seen physicians give a diagnosis of syphilis in children on very insufficient grounds. It should be the mission of physicians to oppose such a condition of affairs, and to insist upon opposition being made to these false and ill-grounded opinions —this, of course, only being possible by the diffusion of a more complete knowledge of the subject than is usually held by the average practitioner. An incorrect diagnosis in syphilis may do an irreparable injustice, not merely to the individual, but to others concerned, and thus make many a life unhappy.

Fig. 1.

THICKENED
PERIOSTEUM

Fig. 2.

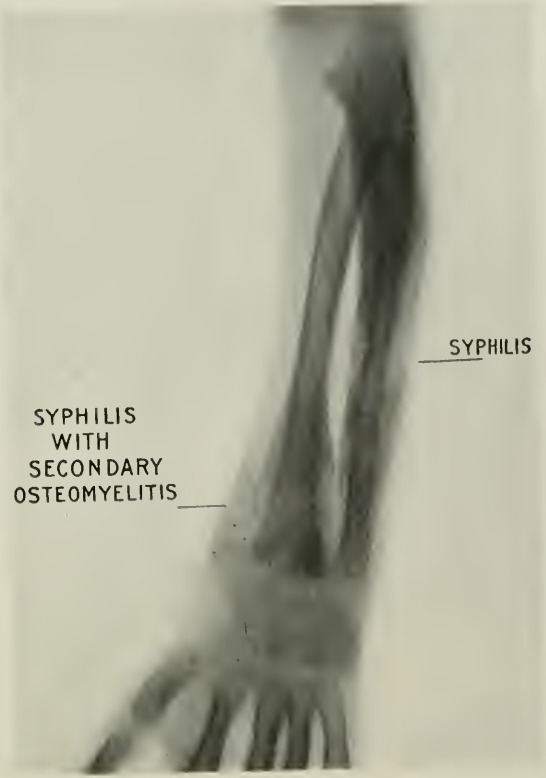
SYPHILIS
SYPHILIS
WITH
SECONDARY
OSTEOMYELITIS

Fig. 3.

Fig. 4.

As an instance of such mistakes I may mention a few striking examples. A consultation of a number of physicians was held over a young child, and the unanimous opinion given that the child had syphilis, with the necessary corollary that it had acquired its syphilis from its parents. In this case one lesion was relied upon for the diagnosis—namely, an ulcerated dactylitis of one of the fingers. This opinion brought grief and unhappiness to two perfectly innocent people: the mother taking it for granted that the father was at fault, because she had been told that acquired syphilis can be apparently cured and yet show itself years after in a syphilitic child without the mother necessarily showing any symptoms. On the father's side was the feeling of absolute grief and dejection that what he knew was not true was believed by wife and physicians, and that under these circumstances his own denial would not be held admissible before the court wherein he was charged. This very child was later proved to have had a tubercular dactylitis and not to be a syphilitic.

It is now well known that a certain diagnosis cannot be made by mere surface examination of such lesions, and that it is by other means, such as the Röntgen ray, that a man in such a position can be cleared or acquitted. Why should a child who has the misfortune to be infected by the bacillus of tuberculosis, the results of which may follow him all his life and yet have his good name remain, be stamped with one of the so-called venereal diseases and looked upon with suspicion throughout childhood, other children being made to avoid him for fear of contagion, and eventually the question of the marriage state being brought up to him in his adult life and the fear of infecting an innocent women keeping him from marrying.

Another instance was that of a girl three years old who, on account of some unusual lesions of the skin—lesions not usually occurring, and, therefore, as is almost always the case, representing in the minds of physicians syphilis—was treated for syphilis and had the stigma of syphilis cast upon her. Yet if a little more care had been taken to use all our instruments of precision in determining the nature of the disease, she would at once have been absolutely cleared of the stigma of syphilis. The Röntgen ray disclosed no syphilitic lesions, but the tuberculin test showed at once that the lesions of the skin were produced by the bacillus of tubercle, and

the child dying later, the post-mortem showed advanced tuberculosis in the region of the ileocæcal valve and appendix.

Such instances as these should warn us not to jump hastily at the conclusion that syphilis is present when it is not, and should impress upon us that with more knowledge, and with a wider spread acquaintance with the best means at our command for making a diagnosis, we may become more charitable in our opinions delivered to the people themselves and to their friends. We should protect the public from the stigma of syphilis as well as oppose the spread of the syphilitic organism.

Part of the mission of this society, as I look at it, is not only to attack the subject from a moral point of view, and endeavor to cure this disease when present by the most approved methods, but to disseminate the knowledge of what these diseases really are, and to impress upon the public that no individual should be branded with the stigma of gonorrhœa as originating from an improper source without first investigating every side of the case to see if some of the rarer forms of infection, such as through young babies, be eliminated. It is also of extreme importance that we should not hastily brand an individual with syphilis, and that the opinion that syphilis is present should be given with caution and only when it is absolutely proved that some other organism has not infected the individual in such an obscure way as to simulate syphilis.

I assure you that it is only by a combination of forces—scientific as well as moral—only by team work in this subject that these infections can be gotten rid of, and the public protected in its ease of mind as well as in its physical condition.

The power of knowledge should be used to destroy gonorrhœa and syphilis to the utmost, without flinching, vigorously, unrelentingly, far beyond what is necessary in any other infection which afflicts the human race. In so doing the greatest good can be done to the people of the present and to the citizens of the future.

I have spoken with some fervor, but surely, in consideration of the far-reaching scope and importance of subject, none too strongly.

From my point of view I would that I had the ability to double the strength of my argument and to drive it home against that great and serious opponent and antithesis of Knowledge, that stronghold and support of venereal diseases—IGNORANCE.

THE ESSENTIALS OF SCIENTIFIC INFANT FEEDING

BY GODFREY R. PISEK, M.D.

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When it was first attempted to place artificial infant feeding upon a scientific foundation certain supposed basic principles were formulated; these were accepted by many leaders and teachers without question and will be found to-day in a large number of text-books and journal articles, although they are certainly incorrect. Among these principles will be found the following: Milk of all animals is alike except in percentage composition and in reaction. Human milk is alkaline and cow's milk is acid in reaction. In modifying cow's milk to make it like human milk in composition the amount of proteids is to be reduced, the quantity of sugar increased, and an alkali added to change the reaction from acid to alkaline. As there are different kinds of human milk no one modification of cow's milk will suit all infants, but by a little trying and experimenting the right combination for each infant can be found.

The teaching was very plain that human milk was being made from cow's milk, and the inference to be drawn was that only a comparatively few modifications would be necessary. Infant feeding seemed to be as easy as anything could be and to rest on a few problems in mathematics. But when it came to applying these principles it was found that they did not work in practice as easily as they read. The babies did not act as if they were taking human milk and clinically the teachings had to be modified. Thus practice has gradually diverged from theory until they are now widely separated.

Quite a few men never accepted the principles above mentioned, and as a result of their objections a new turn was given to the theory of modifying milk. It was conceded that the theory described had been wrong, but it was claimed that now it was

known that the milks of different species of animals are unlike because of the different proportions of caseinogen and lactalbumin present, and the whey and cream mixtures came to be advocated as the proper substitute for mother's milk. As a matter of fact, it has been proved beyond a shadow of doubt that the caseinogen of the various species of animals is not the same, and also that strictly speaking there is no such body as caseinogen, this substance being a combination of casein and lime. However, in practice these mixtures do not conform to theory and the working directions given with them when carefully studied show that a decidedly different treatment of the food is made than is stated.

It is not uncommon to find text-books laying down both of these theories, which contradict each other, and then following neither in practice; and it is not at all surprising that the general practitioner finds himself utterly at a loss to grasp the situation.

The purpose of this article is not to point out the inconsistencies, errors, and fallacies from which these theories of infant feeding were constructed, but to show what are really the scientific foundations or essentials of successful infant feeding. Instead of laying down laws an attempt will be made to show from phenomena which are familiar to all what the laws of infant feeding are and allow the reader to form his own conclusions.

The maternal nutrition of the infant commences at conception. The developing ovum at first absorbs nutriment from the fluids by which it is surrounded, but as the organism develops it attaches itself to the wall of the uterus and through it obtains food. As organization becomes more complex the placenta is formed, and gradually the circulation of the blood is established. When birth occurs a sudden change in the method of obtaining food takes place. The mother now supplies it from the breasts instead of through the placenta. At first she secretes colostrum, but this is soon displaced by milk which she supplies until teeth are cut and the infant is ready for solid food. During the time the infant is at the breast its digestive organs are slowly assuming the form of those of the adult and are gradually developing their functions, as is shown by the ability to take solid food a little at a time. It must strike anyone who thinks of the matter as remarkable that one food, milk, should be suitable for the undeveloped stomach of the very young

infant and also for the stomach that is strong enough to begin to digest solid food. Upon stopping to consider, it will be seen that up to the time that the infant is provided with a digestive apparatus that will enable it to live upon the food ordinarily used by the parents, Nature supplies the food. During the time the digestive organs are developing Nature sees that the infant has food that is specially suited to it. A glance around shows that this same rule holds good with the lower animals. Is it not then likely that there is something peculiar about milk that makes Nature supply it to so many young animals? The young animals grow more after they cease to be supplied with milk than they did when taking it, so, as a food substance, milk cannot be superior in nutritive value to many other articles of diet which, as everyone knows, are unsuitable for young infants. It is self evident, then, that before any scientific feeding of infants can be done, we must know what there is about milk that is different from other foods which are just as nutritious, that makes it suitable for infants and young animals.

The infant's stomach when developed will secrete pepsin and hydrochloric acid, and, as its function is developed gradually, the quantity and strength of these secretions must become greater as the infant grows. During the first few days after birth little pepsin is secreted by calves and presumably also by infants. At this time the udder and breasts secrete colostrum. When the milk supply is established the stomach secretes pepsin or rennet which many believe to be identical. This ferment immediately acts upon the milk and changes it from a fluid into a more or less solid mass, but has little digestive action upon it, digestion being intestinal. When dilute hydrochloric acid is added to milk to which pepsin has previously been added a curious change takes place. The solids become firmer and so altered chemically that the pepsin will commence to digest the proteids of the milk. When pepsin and hydrochloric acid are added to the milk of different species of animals it is seen that the different milks do not act alike. Cow's, goat's, and sheep's milk turn into a solid mass; human milk changes into a mass of small particles, while ass's milk forms a soft fluid jelly. Common experience has taught that Nature provides organs of special structure to perform special functions. And when we

see blood clot in wounds we naturally think what a wonderfully wise provision to prevent bleeding! When we come to examine the process of digestion in the different kinds of animals it is found that they do not all perform this function in the same manner. The milks that behave in the same manner when acted upon by pepsin and acid, are supplied by animals in whom the digestive process is the same, and it will be in line with all other conclusions if we believe that the differences in the solids produced in milk by the action of the digestive juices are wisely provided by Nature so as to make the milks suit the different kinds of digestive processes.

It would seem reasonable to conclude that during the period the digestive organs are developing, Nature intended to provide a food that would furnish more work as the digestive secretions became stronger, and left it to the stomach to determine by the action of its secretions on the mother's milk just how solid the food should be. Doubtless Nature could have had the digestive organs developed at birth, as well as the lungs, but she did not, and it would be difficult to conceive a more admirable plan of providing food that should be just suited to the growing digestive organs than to supply a fluid food which has the property of being changed into a solid food of varying digestibility depending upon the strength of the gastric secretions. As stomachs of all animals are not alike it would be surprising if milks did not have differing digestive properties.

The reason why foods that do not contain fresh milk are not successful in the long run for feeding infants, is that they do not have the property of adapting themselves to the changing stomach and keeping it properly at work. Unmodified cow's milk disagrees with most young infants because of the character of the solid formed from it when it comes in contact with the pepsin of the stomach.

All methods of modifying cow's milk that have been proposed are based on altering the character of the solid formed in the stomach, no matter how this fact may be disguised, as by "feeding low proteids," "split proteids," "overcoming the acidity of the cow's milk," "peptonizing," "decalcifying the milk," "citrate of soda feeding," or "using cereal diluents." Feeding low proteids consists in reducing the amount of the solid or curd-forming por-

tion of the milk. Using split proteids means removing all or part of the curd-forming ingredient of the milk. In *practice,* "overcoming the acidity of milk," means neutralizing the acid of the stomach or making the milk alkaline so that neither the pepsin nor acid can act upon it and form a curd, for the rules of the text-books often call for the addition of more than enough alkali or antacid than is necessary to neutralize the milk if it is sour or than is necessary to neutralize as much gastric juice of an adult as there are ounces of food. Peptonizing, decalcifying or adding citrate of soda to milk have as their object the altering of the milk so that no solid will form in the stomach. The addition of the cereal diluents is to soften the curd mechanically.

Having pointed out the process of nutrition during infancy and the effect of the different modifications of cow's milk, it will be necessary to allude to the food requirements for actual growth. The true growth of the body—the formation of muscle and bone—is absolutely dependent on the proteid obtained from the food, which is represented by lean meat, eggs, curd of milk, gluten of cereal, etc. If this element of the food is deficient, a weakened constitution will result, although the infant may gain in weight rapidly if there is sufficient sugar present. If there is enough proteid but insufficient sugar and fat in the food, stunting will follow because the proteid cannot be stored up as new tissue but must be used for the current needs of the body, which would normally be met by the sugar and fat. It will, therefore, be easy to see the importance of knowing approximately the composition of the food. Many times the condition of an infant can readily be accounted for by determining the composition of the food which it has been taking during a certain period.

It may safely be set down that the essentials of successful infant feeding are as follows: (1) To supply in the food enough fats, proteids, sugars, or carbohydrates, mineral matter, and water to allow of normal growth. (2) That clean fresh milk be used as the basis of the food, as it is the only available food that has the property of changing into a solid when acted upon by the gastric secretions, and to furnish proper work for the developing stomach. (3) That the modifications of the milk which have for their purpose the modification or altering of its curding properties, or reaction

with pepsin and acid, interferes with the normal digestive process as little as possible.

Just how these essential requirements may be met in all cases cannot be learned from a book. No two infants are exactly alike and then, when the physician is called to see a child, it is usually suffering from disturbed digestion or malnutrition. In such cases anything that will tide the infant over the period during which its digestive apparatus is being brought back to its normal condition may be used. But the mistake should not be made of supposing that a food is the proper one because it succeeds for the time being. It will not be satisfactory for any length of time, unless it meets the above requirements.

There is one point that should be borne in mind, and that is the wonderful effect the appetite has on digestion. Sometimes the mere change of the taste of the food without changing its percentage of composition will bring about good digestion, and a food that was refused or taken with indifference may be clamored for when one form of gruel diluent is replaced by another, or cane sugar used instead of milk sugar. The success of many of the best feeders is due to their ability to meet the foregoing essentials by a variety of methods. Experience alone cannot make a good infant feeder, for oftentimes the more experience one has the more confused he becomes, unless he learns to discern between the essential and non-essentials. A great advance will be made if the profession will believe that infant feeding is not such a complicated subject as a perusal of many of the text-books and articles on the subject would imply, and take courage and learn how the really few essentials may be covered. Each case treated will then be an education which will make the next one easier to manage, for one soon comes to recognize the cause of many of the difficulties encountered, and sets out to remove it instead of vainly trying to make a special mother's milk for the infant. The improved methods of preparing food that are now available make the practice of scientific feeding in the home a simple matter.

Neurology

INSANITY IN THE AGED*

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One great difficulty in studying mental disease in the aged is in distinguishing pathological symptoms from normal changes. Completely established senility is easily recognized, but its onset is often insidious, and mental ability and disability may be so combined as to make a decision concerning the real mental state very difficult. Indeed, if a man is to be regarded as mature only as long as he retains all his faculties at the highest point of efficiency, most of us begin to be senile in middle life. Thus deficiency of memory, the common first symptom of normal mental dissolution, begins, as a rule, at middle age and while the man is still growing in wisdom and judgment. Loss of power of recollection shows itself first concerning isolated facts having no mental association with others, *e.g.*, proper names, faces, and arbitrary dates and other numbers. At the same time mere sensory impressions cease to be as firmly imbedded in the mind as before. On the other hand, the recollection of occurrences of early life is retained very late in normal old age. Nearly allied to memory is the power to fix the attention closely and for a long time on new things, and in middle life this begins to be more difficult for the average man. Hence, new things, new subjects of thought along unusual lines, are difficult to learn. Memory and power of fixed attention are closely connected with the entire mental life, and when they are lost or even greatly impaired dementia necessarily results.

One of the most positive signs of senility is a numbing of the emotions. This is normal, unless extreme. For an old man to feel

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less strongly than the young; for him to look with more or less indifference toward death, and not to feel so acutely as formerly ills coming either to himself or those nearest to him, is not a sign of disease. In some degree it is a result of education in life, but much more largely is it a physiological emotional anæsthesia. When it is caused by great increase in self-centeredness and egotism it is pathological. Along with the lessening of the higher emotional feelings there may be, and it is pathological if uncontrollable, a temporary recrudescence of the lower animal passions, and an old man may fancy himself in love or loved by some young woman, may become the victim of designing adventuresses, or may commit or attempt sexual crimes on children. Some old men convicted of such crimes are entirely irresponsible for their acts, and are really suffering from senile dementia. Thus, one old gentleman, who had been a kind father and of good habits, rapidly deteriorated in memory and judgment, but was not regarded as seriously ill till he began to make violent love to his daughter. Pathological change in disposition and feeling based upon the sexual instinct is an occasional cause of divorce suits. A man happily married for many years may, without showing at the beginning any other marked sign of mental disturbance, develop delusions of marital infidelity and in consequence be suspicious of his wife and accuse her falsely. He may be so matter of fact in his statements and so plausible as to convince others of the correctness of his belief. I am not speaking of mania in which insanity is patent, but of beginning mental unsoundness in which cunning and apparent shrewdness throw the observer entirely off guard. Sometimes sexual excitement and delusions of suspicion last only a short time and are rapidly followed by dementia. Thus, a widower, seventy years old, whose previous health, both mental and physical, had been good, was seized with an apoplectic attack without palsy, followed by a period of mental confusion and excitement, lasting several days. After the confusion had passed off he became very suspicious of his daughter and said she was talking ill of him and wanted to get him out of the way. At the same time he determined to marry a woman he scarcely knew, and who before the stroke he would never have regarded as a proper person to be his wife, and sung her praises constantly. He resented very much being restrained from seeing her, and threat-

ened all kinds of evil against the members of his family for not allowing him to do as he wished. At first his judgment concerning matters foreign to his delusions was but slightly impaired, and in casual conversation no one would have suspected there was anything the matter with him. After several weeks he began rapidly to degenerate, and by the end of three months was much too demented to have any delusions.

Normal old age may be happy or querulous. External circumstances are not the only factors which determine the predominant emotional tone. It depends much more upon the nature of the man himself than upon whether he has had worldly success or not or whether his burden of sorrow has been light or heavy. Here, as everywhere, there is no distinct line dividing the normal from the pathological. The safest rule is to regard as pathological that which is not the natural outcome and continuance of the emotional habit of the past life. For a man who has always been easy-going, careless of trouble and able to throw off grief to become captious and quarrelsome; for another who has been serious minded, prone to take life heavily and to complain much, to become sillily happy, are alike pathological. It is not uncommon, however,—we see it every day,—for men who have been hard fighters in early and mature life to soften later, but long before any signs of dementia appear. It is customary to assume that this change in disposition is due to having learned wisdom: it is more frequently caused by emotional anæsthesia.

Impairment of intellectual judgment is one of the most serious indications of senility. At first intellectual processes are simply carried on more slowly. The result is just as correct as before. At the same time or later the man begins to be less self-reliant. He who never asked advice now begins to lean on others. He who never doubted himself is now never quite sure. The reverse also occurs, and the man who was easily led becomes obstinate, self-willed, opinionated. Later, when dementia is established, judgment fails entirely.

As a rule, mental and physical old age run along together, but the old saying, "A man is as old as his arteries," is not always true. Some men grow old in mind, become senile, and, indeed, even reach dementia, at an early age, and while the arteries are still soft

and free from disease. I do not refer to dementia secondary to some acute insanity, which may occur at any age, but to primary pre-senile dementia, which seems to be dependent upon an inherent weakness in the man himself, an inability to withstand the stress and strain of life beyond a point far below that of the average man. On the other hand, there may be marked cerebral arterial sclerosis without any mental failure. One of the most distinguished professional men America has produced died when fifty-six years old. He had marked heart disease, and the larger arteries of the brain were so hard that, separated from the cerebral substance, they supported themselves like the branches of a tree; yet he showed no mental defect even a few days before death. Even in extreme old age with arterial sclerosis mental power may be retained to a surprising degree. For example, a woman who died in her ninety-first year, retained good judgment, interest in her family and in affairs in general, and received a great deal of rational pleasure out of life. The only mental defect she showed was some loss of memory. In the cases in which the small arteries are so much diseased that they are occluded by the thickening of their walls or large numbers of small thrombi form and the cortex and white matter atrophy in consequence of insufficient blood supply, of course, the dementia is the direct result of the arterial disease. This had not occurred in either of the cases cited above. Severe as the atheroma was, the small arteries were all, as far as could be determined, fairly patulous and there was no cortical atrophy. It is probable that senile dementia occurring in people whose arteries are not extremely diseased is sometimes caused by the failure of the excretory organs of the body to fulfill properly their functions. It is probable also that sometimes a failure or perturbation of function of some of the ductless glands may be a cause.

The differential diagnosis of normal and pathological old age is often difficult. There is no clearly marked dividing line, and often health and disease are so combined,—the patient seems so bright and natural in some respects, so dull and perverted in others, —that a decision is almost impossible unless the man be watched and studied for a long time. The matter is of more legal than medical interest, because there is no cure for old age except that which cures all ills. In practical life the question is most frequently

brought up in court in will cases, and is fought over with hypothetical questions, which may or may not contain all the facts of the case, and statements of lay witnesses that the patient did this, that, or the other thing, each act being held to prove sanity or insanity. It is often forgotten that insane men may do many sane things. Lay witnesses are prone to forget that the discovery of the motives of isolated acts is not always easy, and that sane men may do unusual things. Mere eccentricity, which is often brought up in court, is no evidence of insanity. A safe medical judgment as a rule can only be formed by examining all the evidence, and, if possible, studying the man himself. Too often in examining patients so much attention is paid to what they say that a study of their behavior is neglected. The latter is quite as important as the former. A man's conduct is as important as his words in determining his mental state. The elderly man who, having been careful of his appearance, methodical in his habits, accurate in his business affairs and affectionate in his family relations, becomes, without physical cause, slovenly, careless, and without regard for the welfare of his family, is rapidly approaching, if he has not already reached, the beginning stage of mental senility. If to these there be added marked forgetfulness of recent events and distinct loss of affection for those nearest to him, he has gone one step further along. If, in addition, or in place of these, there arise causeless hate and delusions of suspicion or grandeur, boisterous garrulity and silly vanity, or, as may happen, melancholia, with delusions of self-accusation, he surely is over the boundary separating the sane from the insane. The last stage of all needs no physician to make a diagnosis; mental functions all cease, and he lives by his spinal cord and basal ganglia alone.

The true insanities of old age may be classified clinically (it is still impossible to make a consistent scientific classification), as follows: Simple chronic dementia, acute dementia, chronic dementia with delusions, including senile paranoia; post-apoplectic dementia, post-traumatic insanity, mania and melancholia.

Simple chronic dementia is the most common. It comes on slowly and is characterized by increasing loss of memory and judgment, with silly happiness alternating with occasional outbursts of querulousness or even impotent rage, with marked egotism and only

vague and passing delusions and hallucinations. Though the trend is steadily downward, careful observation will show that progress is intermittent and at times flashes of the old intelligence recur. Usually there are transitory attacks of tears and childish woe. Some patients maintain for months and even longer a state of mild mental and emotional excitement. They are always a little over-excited and self-satisfied, garrulous and opinionated. They are continually wanting to do things which they are not competent to do and to concern themselves with the affairs of other people. The prodromal period may extend over many months, and even after dementia has become complete physical life may linger a long time.

Acute dementia has many exciting causes. Almost any acute disease occurring in the aged may be the beginning of a mental breakdown starting with delirious excitement and ending with dementia. Such a result is very prone to follow a febrile attack from any cause. Sometimes it is impossible to tell at the beginning of the illness whether the mental symptoms are those simply of transitory delirium or whether it is the commencement of dementia. I have tried to discover the relative liability of fever to produce transitory delirium in old people as compared with children, but have not enough data to draw positive conclusions. So far as my experience permits me to judge, I believe that fever causes delirium in the old as frequently as in children, and, while delirium from simple fever in the child is of no very serious import unless it is long continued, in the aged it is often a danger signal of possible later dementia. Autointoxication from the intestines, or, at any rate, relieved by purgation and regulation of diet, may produce a state of hallucination and delirium which may make one fear the onset of a serious dementia. Thus I saw recently a lady seventy-six years old, who, barring a slight stroke which she had had some months before and which had left a persistent feeling of numbness in the left arm and leg, without any palsy or objective anæsthesia, had always been well till a couple of weeks before I saw her, when she began to have visual hallucinations of men and women always appearing on the left side, sometimes standing, sometimes sitting, sometimes making commonplace remarks, and sometimes crying fire and murder. The hallucinations were not constant, but came and went, and appeared most frequently at night. Several times she

was so scared by them that she jumped out of bed and tried to run away. When I saw her she was quite sure of their reality, but she was so normal in other respects, talked so naturally and seemed to have such a sane view of things in general, her moral sense was so acute, her memory for recent events so good, that I gave it as my opinion that probably it was a transitory disturbance. Free purgation and regulation of diet was followed in a couple of days by the disappearance of the hallucinations and a few days later by the realization that they were imaginary and not real. She is now well. The chronic stage of acute dementia is precisely like that of dementia chronic from the onset, and, indeed, cannot be distinguished from it.

Frequent as post-paralytic dementia is it is not an inevitable consequence of apoplexy. Many old people suffer a severe stroke and have a permanent hemiplegia without any serious mental symptoms whatever. Whether dementia follows or not depends on how severe is the general cerebral arterial sclerosis, how extensive the thrombosis (for the cases are much more frequently thrombotic than hæmorrhagic, the latter almost always ending soon in death) and the escape or involvement of the speech centres. Even aphasia if it be purely motor does not necessarily produce dementia, but when it is sensory, when the visual and auditory centres are destroyed or cut off, in other words, when the patient loses the tool of thought, internal speech, dementia must result.

Aphasia often leads to legal contests, the question being, was the aphasic competent to make a will or sign a contract? Practically the question resolves itself into this, or, rather, these: Did the patient know and fully realize what he wished to do, or what some one else wished him to do, and its consequences? If it is a will, did he know about what he was worth; did he realize who were his natural heirs; was he influenced in making the will by any insane delusions, and did he understand the will itself? This, of course, is a physician's, not a lawyer's, way of putting it. In pure motor aphasia it is relatively easy to discover the patient's mental condition because he can understand all that is said to him, and, though he cannot talk nor probably write, he can indicate his meaning by gesture and pantomine. In sensory aphasia, on the contrary, he cannot understand spoken language or writing, and his speech

is always so mixed that he cannot be understood. Hence, it is safest always to regard him as a dement, which, indeed, he is.

Chronic delusional insanity is not very infrequent among the old. Indeed, there are all types, from simple dementia with only fleeting delusions, through one or two fixed delusions up to the so-called senile paranoia. Sometimes there is apparently true monomania, but, if the patient be carefully examined, in the majority of cases some sign of dementia will be found. Thus, an old woman showed no marked sign except that she was quite sure that her sister, whom she remembered to have seen buried, was really alive, basing her opinions simply on the belief that some one had told her so. She was quiet and well mannered and easily managed. But she had none of the feeling of horror that a normal person would have at the thought of a sister buried alive, had lost all feeling of affection for her relatives, and could, though she had formerly been a strong-willed woman, be led by any designing person.

Much the most interesting of the senile delusionals are the so-called senile paranoiacs. They present extreme egotism. The world is egocentric. They are vain and conceited, persons of great importance, billionaires, popes, cardinals, kings, great inventors, poets, or anything else that seems great to them. They have no moral sense, and admit no law save their own wills. They boast, and strut, and brag. They reason fairly well at first about things not connected with their delusions. The delusions are always fixed and permanent and the patients will argue with more or less perverted logic about their reality and reasonableness. They are always systematized, *i.e.,* are always grouped around some one subject, or there may be one great delusion and few or many minor ones. The patients are always persecuted and always on account of their greatness. Thus, a man seventy-one years old, who had been a skilled mechanic and always thrifty, a short time after being operated on for stone began to be confused and forgetful. Soon he developed the belief that he was the inventor of a machine for making paper bags and had been offered many million dollars for it. At the same time he believed that there was a conspiracy to cheat him out of the money and that his children were concerned in it. He accused his daughter of living an immoral life, saying that a child who would conspire against her father would do anything. He

became so troublesome that he was put in an asylum. After about ten months he escaped out of a third-story window, using the sheets and blankets of his bed for a rope, and walked many miles to this city, doing some odd jobs on the way to get money for food. He worked only a few days when he created a disturbance with a man to whom he was trying to sell his patent, and was sent to the Philadelphia Hospital. When I saw him he was grandiose, cunning, resentful and revengeful. His invention was the greatest the world had ever seen and would bring him millions. The paper bag trust were his enemies and had sent him to the hospital because they did not wish the price of bags cheapened. He wrote long, grandiose letters to lawyers, demanding that they assist him to get him his freedom, and promising large sums for their legal aid. He wrote threatening letters to distinguished men, demanding that they should instantly use all the forces of the government to punish his enemies. The letters were all very rhetorical and grandiose. He differed from a true paranoiac in that in the course of a few months he became distinctly demented.

Another man, sixty-six years old and in whom the development of the disease was very rapid, told me his wife had influenced people against him. She had been a true wife for thirty years, but later began to go out at night. She had joined two clubs, the "cocks" and the "hens." She should have none of his money. Once he saw a woman in the hospital and claimed her as a new wife, saying: "She is a perfect lady right from the wash-tub." He at times heard the voice of Christ, who had given him supernatural power. Five million dollars were waiting for him in New York. He was worth twelve million. At the same time he inveighed against his son, whom he said owed him sixty dollars.

He was conceited, talkative, boastful and resentful. He was disgusted with all of us, and complained of the many petty annoyances to which he alleged he was subjected. We were persons of no importance, and he would show up the conspiracy we were in. In a few months his dementia increased greatly and his delusions disappeared.

Chronic reasoning mania and paranoia run into each other, and in many cases no differential diagnosis can be made. The typical cases are clear enough; the boundary line cases can be put in either

class. It is, however, probably wise to retain the name senile paranoia for those patients who, retaining large but perverted reasoning powers, have fixed and systematized delusions, and to use the term mania to include those whose delusions are not fixed and systematized and in whom there is distinct dementia very early. In the late stage of senile paranoia and mania alike dementia becomes profound.

I doubt if true paresis ever begins in old age. There are undoubted cases of grandiose, senile dementia, but they rarely, if ever, run the course and show the classical physical signs of true paresis. As a rule, the condition is a mere episode in senile dementia.

Post-traumatic dementia is becoming more frequent since the great increase of railroads, trolley cars, machinery, automobiles and other means of hurting the general public. Old people bear shock very ill, and the shock of an accident often does as much injury as the physical hurt. As a rule, the result is simple dementia. Usually the outlook is very bad, but sometimes there is quite marked improvement. Thus, a blacksmith, sixty-five years old, working on a ship, fell through the hatchway and was picked up with a severe concussion of the brain. He soon recovered consciousness, but was much confused for weeks, so that he did not recognize his wife and children. After several months he rapidly improved, and at the end of a year was able to act as a watchman in the ship-yard. He was never able, however, to return to his trade, partly because, as he said, he had lost his nerve.

True acute mania and melancholia are infrequent in old age. When attacks do occur they are usually recurrences, the first attack having occurred years before. I have seen several cases in which a period of many years elapsed between the first and second attacks. I have also seen several other cases in which, though there were many attacks of mania throughout life, life itself did not seem to have been shortened by them. Neither acute mania nor melancholia differ in any way in the aged from the same affection in younger people, except that the outlook for cure is very much poorer.

Curiously enough, though most drunkards die at a relatively early age, true alcoholic insanity sometimes occurs in the aged. Thus, I have seen a man eighty years old (he was Irish) pass

through an attack of acute confusional alcoholic insanity, the last, so far, of a series, the first of which had occurred fifty years before. He left the hospital not much the worse for his experience, and still retaining his racial wit and optimism.

Let me say a few words about the care and treatment of the mentally senile. Some one—it was a lawyer—has said that all bachelors and widowers over sixty-five should have guardians. This is doubtless a somewhat exaggerated statement. In all seriousness, however, many men get into trouble with the law, or lose their money, or bring scandal on their families when they are wholly irresponsible, and should be so regarded and treated. How to prevent such calamities is sometimes a difficult problem. Sometimes the physical disabilities of old age prevent harmful conduct, and it is fortunate when such are present. Sometimes the excitement and violence of the patient are so great that no one can object to sending him to a hospital for the insane. On the other hand, simple dements can be cared for at home, provided they are well enough off to afford proper attendants. When a patient has marked delusions, especially of persecution or grandeur, he should be sent to a hospital at once.

FACIAL DIPLEGIA ASSOCIATED WITH LABIO-GLOSSO-LARYNGEAL PARALYSIS*

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The extraordinary features of the case which I am about to report make it unusually interesting both from the standpoint of the localization and pathogenesis of the affection. Very few cases of this character are to be found in the literature.

Mrs. F., married, aged 35, had for many years frequent attacks of some throat affection. In 1903 had an unusually severe attack of tonsillitis (perhaps diphtheria). After she recovered from it, she had difficulty in talking and singing, but soon improved. In 1904 she was taken with very severe headache lasting several weeks. Then she noticed that her speech became slow and her voice low. Two months later she commenced to stammer and at times could not utter a word. She felt, she said, that "her tongue was tied." In the throat she felt a fulness and pain. Gradually she developed difficulty in swallowing, in masticating and finally lost the power of speaking.

At present her condition is as follows: Total motor aphasia, Cannot sing or even raise her voice. Quite frequently she experiences a difficulty in swallowing all kind of food, but more particularly fluids; they run out of her mouth. Has difficulty of masticating, of moving the food from one side of the mouth to the other. Blowing out a candle and whistling are impossible. Suffers from excessive dryness in her mouth; the lips, she says, stick to her teeth and she gets relief from this sensation by rubbing her gums and palate with her fingers. She protrudes her tongue only very slightly and cannot lift it or turn it towards either side. The tongue is quite thin and appears to be somewhat atrophied. The

* Read and patient exhibited before the Philadelphia Neurological Society October 23, 1906.

uvula does not go up during forced inspiration. There are no fibrillary twitchings of the tongue or of any other muscles. Another striking feature in this case is the immobility of the facial muscles. She is unable to pout her lips, to move them to either side, to show her teeth. Laughing or even smiling is difficult. The forehead is smooth and the skin appears to be tense. She is absolutely unable to wrinkle it or frown. The lower part of the face is hypalgesic. Taste is normal. The pharyngeal reflex is absolutely lost. The electrical reactions show distinct RD for the m. frontalis and for the muscles of the upper half of the face, but only diminished response for the lower half. Faradic contractility is diminished on the entire face. The examination of the eyes shows only this peculiarity, that when she attempts to turn her eyes, they do not remain in the lateral position more than a second; they immediately return to the former position. Moreover, there is some paresis of the right external rectus and inability to close the eyes. She also complains of occasional dyspnœa and palpitation of the heart. The rest of the nervous system presents no deviation from normal. The examination of the larynx shows full power of adduction and abduction of the vocal cords.

Discussion.—The patient does not present the typical facies of bulbar cases. In the latter the mouth is widely open and when the patient commences to laugh, this is continued for an indefinite time, the laughter itself is usually stupid and ridiculous. In my patient these symptoms are absent. The vivacity and mobility of her eyes show the integrity of her intelligence.

Her speech deserves our attention. As is well known, in bulbar palsy there is at first dysarthria and then anarthria. When aphonia is present, one may hear some mild noise when the patient attempts to speak. Moreover, the inability to talk is very rarely complete and when it does occur, there is always an indication in the state of the vocal cords: there is either a paralysis of the constrictors of the glottis (in most cases) or of the abductors. My patient's vocal cords approach and separate, when she attempts to make a sound. Besides, she is absolutely unable to say a word beyond "yes" or "no."

There may be another interpretation of the aphasia in the present case. As mentioned above, the patient is absolutely unable

to articulate the speech or she has lost the power of utterance, but her ability to recall images, to understand all that is said or heard, is intact. As vocalization and formation of words and phrases are performed by the larynx, tongue, lips and movements of the face, it is reasonable to suppose that the lesion in the present case is located in the cortical centre of these organs, viz., in the lower third of the central convolutions of the left hemisphere. The objectionable features in the cortical view of the facio-labio-glosso-laryngeal syndrome are the bilateral involvement of the upper halves of the face and the presence of RD in the muscles.

Another singular feature of the case is the involvement of the superior portion of the facial nerve. In almost every case of the classical bulbar paralysis the paralysis of the tongue and of the larynx is associated with that of the muscles innervated by the inferior facial nerve, but never with the paralysis of the upper facial nerve. There are, however, in the literature a few cases, viz., those of Remak, Bernhardt, Loewenfeld, Eisenlohr, Wachsmuth, Hoppe, Senator, Schaffer, but in all those cases there was also involvement of ocular muscles and of the muscles of the limbs. Facial diplegia associated with glosso-labio-laryngeal syndrome was observed especially in family bulbar paralysis (Charcot, Londe, Brissaud, Fazio). As it is well known, the superior facial nerve which supplies the mm. occipito-frontalis and orbicularis palpebrarum originates from a different nucleus than the inferior. According to Mendel it is situated in the posterior portion of the series of nuclei of the third nerve. Testut and Duval believe it takes its origin from the nucleus of the sixth nerve. In my case the weakness of the external recti make the latter view highly probable.

The last peculiarity of the case is the suppression of saliva. In the majority of cases the patients have the mouth open; there is a considerable discharge of saliva. My patient, on the contrary, presents an unusual dryness of the mouth; the lips adhere to the teeth; rubbing with the fingers over the gums gives her some relief from this uncomfortable feeling. The salivary glands are innervated by the auriculo-temporal branch of the ninth nerve (for the parotid), by the lingual and chordatympani for the submaxillary and sublingual glands. Paralysis of the ninth nerve will explain the reduction of salivary function.

Considering the case from all standpoints, we must conclude that it is one of Poliencephalitis with unusual localizations and unusual manifestations.

As to the nature of the affection we must remember the protracted tonsilitis and particularly the acute attack which the patient claims was diphtheritic. The latter preceded immediately the onset of the bulbar symptoms. The etiological factor therefore appears to be *infectious* in nature.

THE CLINICAL STUDY OF INEBRIETY

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The medical study of inebriety has scarcely begun, although it was discussed thousands of years ago, and practised by the priests in Egypt, who were the doctors of that age. Half a century ago, Dr. Turner startled the profession by urging that inebriety was a disease, which could be cured in special institutions. Ten years afterwards, the first hospital for the cure of inebriety was established at Binghampton. Fourteen years later, it was turned into an insane asylum, and the impression prevailed in medical circles that the treatment of such persons in institutions did not promise good results. This institution in its fourteen years of work treated more than 10,000 persons, and statistics showed that fully 60 per cent. remained temperate ten years after treatment.

The inebriate is always a complex mixture of sanity and insanity. He lives on the border lines, and the public are not able to determine upon which side of the line to place him. When he is intoxicated and delirious there is no doubt; when he is sober his mental weakness is not always clear. If such persons are studied with exactness, and by laboratory methods, the signs of disease are evident.

The great mass of the profession accept the statement of the victim, that he is in full possession of himself, and can stop any moment, and that his trouble is only loss of will power. The efforts to treat these people as diseased are regarded with suspicion. Through all the years of confusional studies, there has been a growing conviction that inebriety must have a physical basis of causation, and that the moral theories do not explain the facts. Patients treated by physicians for other conditions recover. Patients using quack remedies are cured; and the confusion of causes and remedies has constantly increased.

Many medical men who would scorn to recommend patent medi-

cines to their patients, consent and even advise patients to take "gold cure" treatments. The patient on his return is buoyed up with the delirium of exaltation, and profound faith in his cure, which in itself is suspicious. This, with the fact that after years of continuous drinking he could be cured in a few weeks by some unknown means, is not recognized by the physician. Later, the patient will complain of motor troubles, of eye disturbances, then suddenly relapse. If he takes another treatment, the same thing occurs again, and finally the physician becomes skeptical, and believes after all it is a moral disorder. All this is deplorable, and shows how near the physician comes to the border of a new realm of practice, and yet fails to go over and occupy it.

Every one who uses spirits to excess is literally a neurotic and is suffering from some form of psychosis. His nervous system is both functionally and organically diseased and deranged. His brain is impaired and defective; the neuronic vitality and power of control are lowered. He is on the road to pronounced stages of insanity, meaning mental disease in its most prominent forms. How far he has gone down this road is a very interesting question which can only be determined by prolonged clinical and laboratory methods of investigation. Thus, the man who has drunk continuously for years has narcotized and depressed his vital forces, broken up normal nutrition, and started a train of degeneration which may or may not be very prominent, but nevertheless exists.

The man who drinks at intervals to great excess, who is called a periodic drinker, has been shocking his brain at intervals, producing chemical coma, irritation, and delirium. He has been poisoning himself at intervals up to the verge of unconsciousness and death, and because he apparently recovers without any marked symptoms, believes that he is not injured. In reality, intoxication manifested by delirium and stupor is as serious a condition as sunstroke or concussion; and its repetition leaves most serious lesions. Both of these classes are diseased, no matter what their appearance may be or how far they retain their position in society.

Automatically they may continue their accustomed work, and to superficial observers, seem much the same; but when examined carefully, very marked symptoms of degeneration appear. Persons who drink periodically may be practically called "maniacs," and the

desire for alcohol a "morbid impulse" which dominates the mind and body for the time. These are veritable drink storms, resembling epilepsy in the sudden beginning and convulsive movement and abrupt ending. They suggest the culmination of certain causes and sources of irritation, which it is possible to recognize and prevent. Many of these periodical attacks come on unconsciously. In the midst of his active work a man will suddenly rush off and drink, without being able to give the slightest reasons for his conduct. In other instances he will make distinct preparations in business affairs, providing for emergencies that may happen; then go off to some distant place and drink to great excess for a few days, returning sober. If he belongs to the secret and continual drinking class, he will make frivolous excuses and explanations of his absence. On returning, he will explain his feeble condition as due to malaria, rheumatism, or some unusual strain on his constitution. In all circles of business there are persons occupying responsible positions who, for a time, have secret drink storms, go away, and on returning call for a physician. These attacks after a time become more frequent, and the former efforts at concealment are lessened; the patient is bolder, drinking at the club or in his home, but never calling a physician until the attack is practically over. Of course, the progress downward is rapid, and the time soon comes when the physical condition of the patient is so pronounced that there is no question of disease.

There is another class of periodical drinkers who suffer from distinct premonitory symptoms, which they do not appreciate. Thus in one case, insomnia, headache, and disturbance of nutrition will excite the patient, and lead to the most strenuous efforts for relief for days at a time. Then suddenly, the craze for spirits will appear, and these symptoms will pass away. In others, a severe attack of malaria, marked by chills and fever, and unusual prostration, precedes the drink storm. One man has a severe attack of constipation and indigestion, another has rheumatic pains and swollen joints; another is profoundly depressed, or unusually exalted, or manifests intense activity and excellent business judgment; or is stupid and depressed. Others show inordinate appetites for food, smoke excessively, or are extremely irritable and suspicious. These and other most complex and unusual symptoms end in a drink storm.

Strangely enough, few physicians ever recognize the import of these conditions. Perhaps it is owing to their want of knowledge, and the concealment of the patient; but it certainly would seem that exact scientific means at this period, and careful treatment, would constitute a most practical field in medicine.

In many instances the patient realizes the meaning of these symptoms, and seeks help in some institution, or aid from his family physician, who is aware of the meaning of these complex symptoms. In almost every institution where inebriates are treated, a number of patients come before or in the early stages of the drink attack, and are most successfully treated. Persons who have been under treatment before, and appreciate the value of preventive measures, appear often in states of nervous excitement, and receive active treatment, going away restored.

A very prominent officer of the government had drink paroxysms at least two or three times a year. For many years he was able to abort them by going to different institutions, and remaining quiet for a day or two. A physician in New York has four private rooms in his house which are occupied a large part of the time by patrons who come there for help during the drink craze.

In these conditions there are many distinct causes the removal of which would be followed by the subsidence of the drink storm. Thus, in one instance, a man who had drunk for years was, by the advice of the physician, permanently cured by correction of his diet and change of his sleeping-room. Another man in the same condition, living in a noisy part of the city, moved out to a quiet country home, and remained temperate the rest of his life. A third man, after a surgical operation, in which some dead bone was removed, was temperate ever after. These are by no means unusual examples. The periodical drinker is suffering from some central irritation, and an active cause which culminates at intervals in the desire for relief by the use of spirits.

A study and removal of these early causes is the most practical kind of medical treatment. The periodicity of these drink storms, appearing with great uniformity at certain times and in certain places, suggests causes that are yet unstudied. A number of such persons drink at distinct intervals and on no other occasions. In one instance this interval was thirty-four days. In another sixty-eight,

in a third one hundred and twenty days, in a fourth every year on Thanksgiving Day, in others on the Fourth of July, or some other holiday. At no other time was there a desire for spirits, and on this occasion it was so impetuous and impulsive as to be irresistible.

A very prominent clergyman was noted for his yearly absence on the first day of May. He would make no engagements for that time and was always away, although the reasons were not clear to his friends. The family physician discovered that these were drink attacks, and that he went to an obscure place and indulged himself. He persuaded him to go under active treatment, and had him go to bed before the attack, and employ a special nurse. After a week or more of sharp treatment he recovered. The next year the same treatment was resumed. He finally recovered. Had not the family physician discovered and treated the physical nature of his ailment, he would have been lost.

The periodic drinker suffers from a form of insanity which is more accurately described as a psychosis, and in most cases, if treated properly, could be cured. An example of a successful case is the following: A farmer drank to intoxication several times every year, without any special cause or reason. His physician discovered that each drink attack was preceded by great debility and exhaustion, from either physical exertion or mental worry. Whenever these symptoms appeared he was put on large doses of strychnia and atropia. If the former was not well borne, it was replaced by lupulin. Active cathartics and hot baths were given daily. He was carried over this period, making good recovery; and later, when the symptoms returned, the same active treatment was given, combined with bromides at night. This was repeated for several years, until finally complete recovery followed.

The greatest difficulty is to convince the patient and his friends of the gravity of this condition, and the need of long continued care, especially at those times when the drink premonitions are prominent. Each case must very largely be governed by the conditions present. The physician should never forget that the exciting causes of the drink paroxysm are always very complex and variable. They may be in the climate, occupation, nutrition, or other states which can only be discovered by a careful study. A very excellent man became intoxicated on every visit to the seashore. Another could

not visit New York without experiencing a craving for drink. A third always drank when he went to the White Mountains. Keeping away from these places seemed to be a preventive for the desire for drink. In these cases there were certain unknown climatic conditions which produced the desire for spirits. There is a very wide field here for most practical studies, and the physician whose patrons suffer in this way, neglects his duty, if he does not make a special study and use efforts for cure and prevention of these maladies.

The second most prominent class are those who use spirits continuously, and in so-called moderation. The term "moderation" describes a wide variation of opinions, ranging all the way from beer and wine at meals, to strong spirits taken every two or three hours during the day; and it means very little, unless described by the person in exact terms. The common opinion that wine or beer at the table only is a harmless drink, to be compared with tea and coffee, does not prove to be true in our American civilization. Experience shows that drinking at meals in this country is very quickly followed by the excessive use of spirits at other times during the day, and that it is impossible for Americans to confine the use of wines and beers to the table only. Therefore, moderate drinking, in the sense of using only a limited amount of spirits at stated intervals, is practically impossible for any length of time. While there are exceptions to this rule, the so-called moderate and steady drinker is literally the most degenerate and seriously diseased of all inebriates.

Laboratory research with instruments of precision point out very clearly the reasons for this condition. They show that small doses of alcohol, not to exceed half an ounce of pure spirits, taken in any form, depress the senses, and lower the mental activity. Sensory measurements of the sight, hearing, taste, smell, and touch before and after the use of spirits, show that alcohol always depresses and lowers their acuteness. Measurements of muscular power and the fatigue point show a marked diminution of muscular force from the use of alcohol. While the first effect of alcohol may seem to increase the heart's action and muscular power, this is quickly followed by a decrease. The heart that beats 10 or 15 more beats the first few minutes after alcohol is taken, drops down as much below

the normal as it was above. The same is true as regards the muscular power, so that the average of both heart's action and muscular power is immensely depressed and lowered. Measurements of the higher brain forces of memory, of rapidity of thought, of time reaction, of comparisons, all show the same narcotic action and diminished functional activity. These facts are absolute and beyond contradiction. Clinical studies confirm these facts in the lowered vitality and want of control and confusional condition of the organic activities of the brain and nervous system.

Comparative studies of persons who are abstainers, and those who drink regularly, give additional proof. Hospital statistics show that moderate drinkers are more devitalized than others. They have feebler power of resistance to microbic invasions and acute infectious diseases; and also have a higher mortality from pneumonia, typhoid, and other acute inflammatory diseases. In the prognosis of all diseases, the history of having previously used alcohol increases the gravity and possible mortality. Operations on such persons are less successful and more often fatal. These facts are sustained by other studies, showing the derangement in the circulation of the blood in the brain with defective nutrition and deficient oxidization. Also, the action of alcohol, used regularly, is cumulative. The temporary narcotism and depressive effects are continuous, and very quickly merge into organic changes, particularly vasomotor palsies. Thus degeneration and devitalization are more pronounced in the persons who use spirits regularly.

Clinically, these changes are manifest in the delusions and false reasoning and suspicious egotism of such persons. No matter what the condition may be or the experience of the past, they are always confident of good health and ability to stop or control themselves at any time. They never realize any danger or injury from alcohol, but grow more and more delusional concerning their condition. In business circles they become more automatic and mechanical and less capable of adapting themselves to the new conditions. Mental inconsistencies, irregularities, and defects of thought and conduct appear. The more pronounced the sensory degenerations, the feebler the judgment becomes. Lower down in the scale, all these conditions become veritable delusions, hallucinations, states of dementia, and mania.

In many moderate drinkers, the first sign of insanity is suspicion of persecution, particularly of those who are nearest to them, with feeble consciousness of duty and responsibility; or the patient may become excessively credulous and show extraordinary childishness. All these are premonitory hints of the adumbra shadows of the final eclipse. It is a most practical question whether or not these persons can be cured by active medical treatment.

Formerly, moderate drinking was considered a sort of ideal condition, and no thought of medical treatment was entertained. Within a very recent period the moderate drinkers have been startled at the suspicions of capitalists concerning their mental integrity. They are discharged, and forced out of all positions of trust and responsibility with increasing frequency. Bitter experience proves their incompetency and inability to do work requiring clear exact thought. This brings them under medical notice. The family physician is appealed to, and an effort to become an abstainer is made. Their own assertions that they can do so at will fail, and with half faith and half doubt they appeal to the physician.

The following example illustrates these conditions:

A., a banker, who used spirits in so-called moderation for a long time, was forced to resign his position or become a total abstainer. The effort to stop was followed by so much discomfort that he gave it up. A second and third trial resulted in failure; then he came under the charge of the physician. He was taken to a sanitarium and treated in a most elaborate way for nerve and brain exhaustion, and finally recovered. Then went home and resumed his position. He relapsed and went under the care of the physician again. He was given large doses of quassia several times a day, together with baths, lupulin at night, and saline cathartics. He recovered and went back to business, and since then has continued a temperate man, but goes under the physician's care at intervals for nervousness.

A second instance was that of a manufacturer who, on account of a slight injury, called in a physician. He was a moderate drinker. The physician discovered sclerosis of the liver, with heart feebleness and nutrient perversions. He was persuaded to abstain and take an active course of treatment, beginning with strong solutions of quassia, until the drink craze died out, and then following with strychnine and atropia in small doses, together with other means

which were continued for many months. The result was complete recovery. Had the physician failed to realize his condition and treated him temporarily, the patient would have been lost.

Often moderate drinkers should take institutional treatment in which a change of surroundings and removal of active exciting causes can be obtained. Here the delusional conditions of the value of alcohol can be overcome, and the necessity for an entire change of living made apparent. Then the patient can return and continue the general principles of treatment under the care of the family physician. The craze for spirits is less maniacal and impulsive than that of the periodic drinker; but the prostration and physical depression following the withdrawal of spirits is more profound. States of neurasthenia, and muscular fatigue with neuralgias and local derangements, together with profound anæmia and hyperæmia, are common.

The removal of spirits uncovers the harm which has been done. The oft-repeated statement of the patient, that he feels so much worse since giving up the spirits, is suggestive of the need of medical help. This is analogous to the drug-taker's condition, who, when the drug is removed, suffers from states of nerve irritation and exhaustion which are very acute for the time; and unless he is restrained he resumes the drug for the comfort and rest it brings him.

The inebriate who has reached chronic stages, or who after long years of continuous or periodic drinking has become manifestly degenerate, will appeal to the physician for relief from some particular trouble, usually of digestion. He will come complaining of rheumatism, meaning pain and swelling at the lower extremities, or pain over the region of the liver, with shortness of breath from exertion and general weakness. Often the physician will confirm the patient's diagnosis of these conditions, and enter upon a course of treatment for these states, incidentally advising him to diminish or stop the use of spirits. If the medical man makes a careful study of his case he will find neuritis, cirrhosis, and either atrophy or hypertrophy of the heart, together with mental symptoms of alcoholic psychosis. If he realizes the actual condition, the treatment will be different. Such persons are curable to a certain extent, and in many cases marked restoration has followed active treatment. Usually the physician, after a few weeks of therapeutic experiments,

gives up the case, and the quack or the patent medicine man comes upon the field. The following is an example:

A merchant, who drank at irregular intervals to excess, called in his family physician to what seemed an acute articular rheumatism. A few weeks' treatment failed. The patient was permitted to use wine for its tonic properties. Another physician was called; his diagnosis was very elaborate, but neither of them recognized alcohol as influential in the cause. Later, he was discharged as having failed. A travelling quack came in charge, and he cut off the spirits at once, and insisted on using a magic water, which probably contained some simple salines. This, with baths daily, into which some peculiar drug was placed, constituted the entire treatment. A full recovery followed, and the quack was raised to great honor. Later the patient went to business and resumed his use of alcohol. The troubles returned and a new physician was called, who recognized the active cause, and insisted on total abstinence, together with free elimination and hydropathic treatment. The man recovered, the physician was retained, and continued the medical care and watchfulness, impressing upon the patient the danger of alcohol and the necessity for taking extraordinary care of himself.

These cases are by no means uncommon. The fault is in not recognizing the influence of alcohol. A very large number of so-called rheumatic cases in persons who acknowledge to be drinkers are clearly alcoholic neuritis; and other cases complaining of great exhaustion and insomnia are toxæmic. Disturbances of nutrition, with degenerative liver and stomach, are all referable to this one cause. Of course, there may be and in a large proportion of cases are, some antecedent causes or predisposing conditions favoring the use of alcohol.

We are all familiar with the inebriate in the terminal stages, as seen in the lower courts, charitable institutions, and slums of large cities. The later teachings of science show that a large proportion of these cases could have been successfully treated in the early stages, and prevented from becoming a burden to the community and their friends. The saddest part of this truth is, that many of these people are in our neighborhood, family circles, even by our firesides, and are our most valued friends and patrons. We, as medical men, are indifferent, and fail to recognize the damage

from the narcotic effects of alcohol and the conditions which provoke its use. The poor victim goes on from bad to worse, and we stand by oblivious, until pronounced signs of disease come on, and even then, our treatment is half-hearted, stupid, blundering. The quack comes along and gives relief, which should have come from us long ago by rational common-sense means, and not by mysterious mystical measures.

The question of cure and prevention is as definite as in typhoid fever or any other disease; but it can only come from scientific studies, and by trained physicians. The alcoholic problem is one that threatens in every direction, and calls for medical study, not moral teachings and legislative enactments. Our colleges should train medical men to study this problem from its psycho-physical side, for there is no subject more intensely practical and vital, and it is our duty, as scientific men, to lift the whole subject out of the range of credulity and quackery, into the field of exact science.

ON SOME OF THE MORE RECENT LITERATURE OF TABES DORSALIS: PATHOLOGY AND ETIOLOGY

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As might naturally be expected, the researches on this disease during the past few years (1904-1906) have been very numerous. During this time over 500 studies of greater or lesser value have appeared. These have considered the general features of the disease, the etiology, the symptomatology, pathology, diagnosis, and treatment. Some of those bearing on pathology and etiology will be here considered.

The only general monograph of importance that has come to our notice is that of Erb.[1] While Erb's paper is not as extensive as many others, it possesses all of the strength and individuality of that well known clinician's character. He takes much comfort in the steadily increasing concordance of opinion relative to the syphilitic etiology of the disease, insisted upon by him at least thirty years ago. He is ready to follow the lead of the French pathologist, Nageotte, with reference to the pathological lesions of the disease, while not entirely ready to abandon the idea of its being a system disease. As to the importance of the impairment of the efferent sensory fibres in the interpretation of the ataxia, Erb is willing to accede to the newer interpretations, endeavoring at the same time to retain some of the older conceptions predicating disease of coördinating tracts. The hopefulness with which he regards the subject of treatment is of particular moment at this time, when so many methods for the amelioration of the discomforts of the condition are being extensively advocated.

That pathological morphology has little new to offer is very

[1] Deutsches Klinik, 1905, vi, 126.

decidedly negatived by the marvellous activity on the part of many students. The use of the newer methods of fibre impregnation of Cajal has given Spielmayer [2] [3] the opportunity to offer some new interpretations. In this study, which he terms preliminary, he shows that the method stains the non-medullated fibres of the gray matter. Control-staining by the Weigert method shows the neuroglia supporting substance in the degenerated posterior columns. There is in the Cajal preparations a disappearance of the coarser fibres as they enter through the posterior roots and terminate in the gray matter and about the nuclei. The pericellular network about the nerve-cells also shows alterations. In Clarke's columns the changes are very pronounced, the cells being very poor in fibrillæ. Similar alterations are noted in the nucleus of Goll, but the nuclei of Burdach are less altered. There is a marked neuroglia increase as determined by the Weigert method. Spielmeyer has attempted to follow the degenerations into the cerebellar cortex, where he reports finding a marked decrease in the fibrillæ, particularly in the dendritic processes of the Purkinje cells. An interstitial neuroglia increase is also present in the cerebellar cortex.

In relation to Spielmeyer's studies on the cerebellar cortex some observations of Taty and Chaumier [4] may be recalled. These authors found in 2 patients suffering from tabes-paresis marked alterations in the Purkinje cells and other ganglion-cells of the cerebellum. In the interpretation it should be remembered that paresis complicated the clinical picture. Weigert's studies [5] should also be commented on. He has for a number of years been studying the cerebellar cortex in tabes and in paresis by means of his neuroglia stains. His findings for both tabes and paresis show a marked increase in the number and thickening of the neuroglia cells in the molecular layer of the cerebellum.

From another point of view Crouzon's later studies [6] [7] are of considerable interest. He has shown, as will be pointed out later

[2] Centralblatt f. Nervenheilkunde, 1905, xvi, p. 187.

[3] Arch. f. Psychiatrie, 1905, xl, 2, p. 389.

[4] Revue Neurologique, 1905, xiii, p. 857.

[5] Neurologisches Centralblatt, 1904, xxiii, 16.

[6] Nouvelle Iconographie de la Salpetrière, 1904, xvii, Jan., Feb.

[7] Revue Neurologique, 1904, xii, p. 796.

in the clinical summary, that the lateral columns may be involved in a certain number (10 per cent.) of the cases of tabes. These patients show a different clinical picture. The involvement of these columns Crouzon believes is to be explained upon the ground of the Marie-Guillain hypothesis, which, as is well known, is founded on a special lymphatic system involvement. The author describes in his first paper the clinical signs in 7 patients, with the pathological findings. In 5 cases the one essential feature beyond the localization of some change in the lateral columns, is the meningeal vessel involvement. In these there is a diffuse disease of the lymphatics. This by extension reaches the posterior and lateral columns, if the pathological process is intense. This author is an adherent of the non-systemic theory of tabes pathology.

That involvement of portions of the cord other than the posterior columns may be seen in tabetic patients, has been recognized for some time, and the coincidence of more than one symptom complex frequently reported. Among these, involvement of the anterior horns is not uncommon. Cruveilhier, in 1850, was one of the earliest to report on this complicating syndrome, and his observations have been extended and confirmed by many others. M. Lapinsky [7a] has given an extensive analysis of the literature of this interesting theme, so rich from the stand-point of clinical observation, yet comparatively meagre from the side of pathological anatomy. He reports in detail the histories of 6 patients suffering from the combination. They showed a more or less uniformity in the history. They all showed signs of tabes and all showed muscular involvement. Arteriosclerosis was present in all, and syphilis was positive in 4 patients. Pathologically, typical posterior sclerosis was observed in all of the cords, and in some, the anterior horn cells were clearly compromised. In 3 of the patients the pyramidal tracts also showed involvement. From the interpretative side Lapinsky discusses the question of the peripheral and central origin of the muscular atrophy, since much controversy has arisen as to this special point. In comparing the symptomatology of the form under consideration with that so fully described by Dejerine as characteristic of the neuritic type of muscular atrophies seen in

[7a] Arch. f. Psychiatrie, 1905, xl, 602.

tabes, Lapinsky comes to the conclusion that his cases were all of the amyotrophic tabetic type and that the lesions in the anterior horns were truly primarily central in their origin. The process is distinguished from a chronic anterior poliomyelitis, both because of a different character of degeneration in the cells, and by reason of the fact that the neuroglia show no pathological alterations.

The great interest that at one time centred about the pathology of the posterior spinal ganglia in tabes has somewhat abated, but has not entirely ceased. Köster [8] has given an interesting monograph. Thomas and Hauser [9] have also contributed to this subject. Köster shows that the changes in the posterior ganglia in tabes are similar in kind to those produced in animals in which section of the posterior roots has been practised. Köster, it is true, showed that the cells of the posterior ganglia degenerate after section of the posterior roots, but they do so much less rapidly than following section of the peripheral processes. His studies show a Wallerian degeneration of the fibres belonging to the cells which at first show cell reaction and later cell degeneration. Köster concludes, with many earlier investigators, that the initial lesion of tabes must involve the posterior roots. The trophic disturbances in these animals experimented upon bear striking resemblances to those of tabes. Thomas and Hauser's investigations of 8 cases of tabes showed that in 2 of the patients there were no changes in the spinal ganglia; in 6, however, there were characteristic lesions found. Cell atrophy, disintegration, and ganglion-cell disappearance were observed. Whether the cell atrophy is primary or secondary the authors do not state, but that the changes in the spinal cord and cells are to be reckoned a factor in the pathology of tabes there is little doubt. This is the view so strongly advocated by the work of Orr and Rows,[10] who give an extended review of Köster's findings. These authors believe that in tabes the fibres are attacked at a point, namely, Obersteiner's ring, where they lose their neurilemma sheaths and enter the cord. The loss of the neurilemma sheaths and the thinness of their myelin sheaths at this point expose the axis cylinders of the fibrils to injurious influences. Orr and Rows,

[8] Zur Physiologie der Spinalganglien u. s. w. Englemann, 1904.
[9] Nouvelle Iconographie de la Salpetrière, 1904, xvii, p. 207.
[10] Brain, Winter, 1904, p. 460.

adopting a modified intoxication hypothesis, assume that the lymph stream possibly contains some toxic substances and thus the degeneration is explainable. The toxin is supposed to cause a primary degeneration of the nerve fibre as it enters the cord, causing first an atrophy of the myelin sheath and secondarily the degeneration of the axis cylinder itself. The degeneration, these authors claim, develops centrifugally, *i.e.*, from the point of injury to the termination of the fibre. They thus oppose Nageotte's teachings, who, as is well known, holds that a transverse neuritis of the posterior root due to an infiltrating sclerosis is responsible for the secondary Wallerian degeneration which, beginning at the distal end, passes to the trophic centres in the ganglia. Inasmuch as Orr and Rows find that the degenerations are irregularly distributed, they assume that there may be abiotrophic conditions of these fibres, or that the syphilitic virus brings about a condition of variable resistance. The conclusions of Edinger are thus seen to be drawn on by these authors to try to account for the variable picture.

It may be seen, therefore, in this very hasty summary of the recent work, that no new positions are gained. The hypotheses of Nageotte, of Marie and Guillain, of Redlich and Obersteiner, all so well summarized by Collins,[11] still stand as yet practically unmodified by their respective adherents, and thus far the newer histological methods of Cajal, of Bielschowsky, Donnagio, and others, have not been applied very extensively to the problem under consideration.

The study of optic atrophy in tabes we leave for the present.

ETIOLOGY.

Concerning the etiology of tabes dorsalis the newer evidence tends more and more to make it "impossible to overlook syphilis as a most important factor." Erb's monograph, already noted, as stated by Schwab,[12] seems to have more or less settled the question, for most observers at any rate. In addition to his monograph, Erb [13] has made a striking increase to his statistical contributions. Since 1896 he has investigated 400 more patients of intelligence

[11] Medical News, vol. lxxxii, pp. 1122-1155.
[12] Interstate Medical Journal, Jan. 1904, Jan. 1905.
[13] Berl. klin. Woch., 1904, xli, Nr. 4.

who have suffered from tabes, so that up to the time of his late contribution he has investigated the histories of 1,100 patients. In these last 400 there were 35 without histories of definite infection; so that in the 1,100 patients, 89.45 per cent. were definitely infected, 26.54 per cent. with chancre alone, and 2.89 per cent. without any reasonable grounds for suspicion. Erb also brings the many cases of infantile tabes and conjugal tabes, etc., to the aid of the doctrine. As a guide to the general prevalence of syphilis in the population he has given some interesting figures.

A few studies bearing on the general question of etiology, however, may be noted as showing that the pros and cons have not been entirely exhausted. Fournier, at a recent meeting of the French Academy, has taken the occasion to go over the entire question in a dogmatic manner, and gives no opportunity for the doubters. His recent statements have been severely attacked by Christian,[14] who asks for proof. This alienist speaks for general paralysis more particularly, yet, including tabes in his discussion, says that there is no doubt that there are some true cases of tabes without syphilis. It therefore cannot be the only essential factor. It may not be out of place to recall the work of Gläser and Matthes, who found so few cases of tabes in genuine syphilitics—less than 1 per cent. These figures have been commented on by Hudovernig and Guzman,[15] who, in a large dermatological clinic in Buda Pest, have gathered statistics. Their conclusions, based on 50 patients with well marked tertiary syphilis, leave little doubt in their minds as to the relationship of syphilis to tabes and general paresis. Fully 59 per cent. of these cases showed these diseases. Twenty-four per cent. had tabes. A close scrutiny of their paper, however, shows that they make out their case much stronger than is warranted. They include only tertiary cases, and assume a number of statements that are purely hypothetical. Thus it cannot be shown by these figures that the studies of Gläser and Matthes have lessened the reasons for argument against the syphilitic origin of tabes dorsalis.[16] Gläser does not hold that there is no relationship between the two affections, but maintains two points: (1) that good statistical inquiries have

[14] Progres Médicale, 1905, Apr. 29.

[15] Neurol. Centralblatt, 1905, Feb. 1.

[16] Fortschritte der Med., xxii, 1904, xii, p. 509; xiii, p. 557.

shown the percentage much lower, 30 to 50 per cent. (25 per cent. in 448 personally observed cases and 25 per cent. in a series of 1,567 cases collected by him), but mainly (2) that were it true that the figures were from 50 to 90 per cent., the mere fact of the antecedent presence of a disease, even in such high percentages, is really no criterion of the etiological relationship, unless it can be shown that the tabes shows the pathological picture of a real syphilitic process. The real question is, How many syphilitics become tabetics? This neither Nonne nor Erb has really answered. Erb has made an estimate of the percentage of syphilis in 10,000 men. He found that in general 21 per cent. became infected with syphilis, although true chancre infections were present in only about 7 to 10 per cent. The relation of the figures to the question at issue is not yet evident. O. Rosenbach [17] is also to be reckoned with those who oppose the syphilitic etiology theory. That Erb has needlessly, or purposely, confused statistics by making an arbitrary division of tabetics of good, better, or best social condition, there seems little doubt, but how far the confusion militates against his argument in general is hard to pick out in Rosenbach's forest of words. Erb's introduction of the incidence of gonorrhœa in his statistics seems inappropriate. Rosenbach contributes very little that is new in the way of actual figures, and his general reputation as a champion controversialist does not help the situation to any great extent.

Light on the situation is coming from another direction, however, which must, to a certain extent, alter the beliefs in what is known as metasyphilis. These lights are coming from the study of coincident lesions of tabes and syphilis, and from the cytological investigation of the cerebrospinal fluid.

Adrian [18] has contributed a very complete and thorough study to the question of the simultaneous appearance of syphilis and of tabes. The presence of old syphilitic lesions not infrequently found in the tabetic has been utilized as a confirmation of the causal relationship by the believers in the Fournier-Erb hypothesis. Synchronous active syphilitic processes in the tabetic, however, must be viewed from another point of view, although even these have

[17] Therap. Monatshefte, 1905, xix, 3, p. 111; 1904, xviii, pp. 111, 197.
[18] Zeitschrift f. klin. Med., 1904, lv, p. 327.

been distorted by the Fournier-Erb followers, and it is this side light that Adrian throws that is of particular interest. He collects the reports of 96 cases of which (1) 16 showed anatomically active signs of syphilis in the central nervous system in tabetics, (2) 15 showed anatomically active signs of syphilis in other organs than the nervous system, and (3) 65 cases which showed during the life of the patient active constitutional signs of syphilis.

It is of interest to note that 12 per cent. of the patients denied any knowledge of the syphilitic infection. From the beginning of the infection to the commencement of the tabes, 24.2 per cent. occurred within five years, 18.2 per cent. between five and ten years, 24.2 per cent. between ten and fifteen years, 12.1 per cent. between fifteen and twenty, 18.2 per cent. between twenty and twenty-five, and 3 per cent. between twenty-five and thirty years. Adrian compares this with the onset of the syphilitic lesion after infection, which he shows for 30 patients occurred in 13.3 per cent. in the first five years, 6.6 per cent. between five and ten, 26.6 per cent. between ten and fifteen, 16.6 per cent. between fifteen and twenty, 6.6 per cent. between twenty and twenty-five, 20 per cent. between twenty-five and thirty, 3.3 per cent. between thirty and thirty-five, 3.3 per cent. between forty and forty-five, 3.3 per cent. between forty-five and fifty. A very interesting table of his shows the time elapsing for the appearance of the syphilitic manifestation *after* the onset of the tabes. Thus, for 44 cases, in 6 the appearance was synchronous, 2 after two months, 2 after six months, 5 after one year, 1 after eighteen months, 3 after two years, 1 after three years, 3 after four years, 2 after five years, 5 after six years, 3 between seven and eight years, 1 after nine years, 1 between ten and fifteen years, 11 after eleven years, 3 after twelve years, 1 after fourteen years, 1 after seventeen years, and 2 after twenty-three years.

Tertiary syphilis, he shows from Fournier's study of 4,409 cases, presents its greatest manifestations within the first five years, and its curve then rapidly falls, whereas the curve for tabes, 325 cases, rises from 14 per cent. in the first five years up to 30 per cent. in the ten to fifteen year after period, and then sinks rapidly. Further, the interesting feature is brought out that antisyphilitic treatment has given excellent results in some cases, so far as the

syphilitic process has been concerned, but has had little or no influence on the tabes. This is an old observation, but its reiteration is of value. The tabes, Adrian thinks, probably has a close relationship to syphilis, but it is not, he says, a metasyphilitic process in the sense of Fournier.

That tertiary syphilis occurring during tabes is much more frequent than has been recognized is further made evident by a study of Dalous in *Les accidents syphilitiques pendant le tabes.*[19] Dalous does not make as complete an analysis as does Schittenhelm, but he reports two new observations, and gives a résumé of 21 other observations, of which it is of interest to note that 9 were ignorant of or denied the initial infection. The individual cases of Gaucher,[20] of Charmeil,[21] Guzman,[22] and Rug[23] add to our statistics bearing on this matter, if they do not contribute new features of importance.

One history, by Guillain and Thaon,[24] is of more than usual interest, showing active syphilitic signs in a hereditary syphilitic of fourteen years of age, with the clinical signs of tabes of hereditary type in combination with encephalic symptoms. He was the son of an alcoholic and later syphilitic father. He was born at term. Up to the age of five he had variable skin eruptions. At the age of six and one-half years he had a right hemiplegia with disturbance of speech. This disappeared in a few months. At the age of seven he had a keratitis of uncertain character. He had a certain laxness of memory and of intelligence. At the age of thirteen he had pain in his legs of a lightning-like character. Ataxia of gait appeared soon afterwards, the legs being thrown to the right and to the left. These phenomena persisted, and in July, 1904, marked difficulty in swallowing appeared, his mental faculties showed marked clouding, and he had frequent attacks of crying and of great rage. On his entrance to La Salpétrière he showed neuralgic-like pains of the lower limbs, Romberg's signs, lost tendon and Achilles reflexes, and an Argyll-Robertson pupil. There was a marked dysarthria, and

[19] Revue de Medicin, 1904, xxiv, p. 71.
[20] Bull. de la Soc. des Hop., Feb. 23, 1905.
[21] Echo med. du Nord, August 24, 1904.
[22] Monatsch. f. prakt. Dermat., 1904, xxxix, p. 703.
[23] Berl. klin. Woch., 1904, xli, Nr. 11.
[24] Revue Neur., 1905, xiii, p. 258.

swallowing was impossible. His mind was clouded and he was apathetic, although there was no loss of orientation.

Under thorough mercurial treatment, persisting six months, he recovered from his ataxia, his Romberg, his pains, and his dysarthria. Abolition of reflexes persists, and there is papillary atrophy of both retinæ. This is certainly an interesting presentation. The exact nature of the lesion can only be surmised, but there is little doubt that the antisyphilitic treatment was of service, and that an acute syphilitic process was responsible in part, at least, for the symptoms observed.

Bearing on the oft-repeated argument that notwithstanding the wide distribution of syphilis among Oriental people there are but few cases of tabes (and general paresis as well), some of the recent literature tends further to strengthen the conviction that other contributory causes must be reckoned with if a full understanding of the tabes-syphilis controversy is to be satisfactorily adjusted.

Papadopoulos[25] has brought out very forcefully that in Asia Minor among the Mohammedans syphilis is very widespread, yet tabes is practically unknown. He emphasizes the fact, already well known, that the absence of alcoholic abuse is an important factor in the interpretation of these results.

Hödlmoser,[26] working in the Bosnian hospitals, in an analysis of over 7,000 patients was able to find only 9 suffering from tabes. He does not say whether these 7,000 had had syphilis or not, so that one is unable to measure the value of his arguments, but he also is inclined to account for the small proportion of tabetics by reason of the small amount of alcoholic abuse among these people (see original).

D'Orsay Hecht,[27] in an exhaustive study on tabes in the negro, reviews the general question so far as this race is concerned. It has come to be regarded, he writes, that colored people are exempt from the disease, and he reviews the evidence carefully on this point. Syphilis among these people, he believes, has increased markedly, especially since their emancipation. Miscegenation is occurring in ever-increasing proportions. All of his cases occurred

[25] Progres Médicale, 1904, xix, 10.
[26] Wien. klin. Rundschau, 1904, xviii, 13, 14, 15.
[27] Am. Jour. Med. Sc., 1903, cxxvi, p. 705.

in mixed strains, and the greater proportion of the reported cases in negroes occur in those of mixed blood. Hecht believes that Aryan blood is necessary for the production of tabes, and that the disease is probably more common than is supposed.

To those who contest the syphilitic origin of tabes and yet have little new to offer may be added a few who report instances of the development of tabes following some other factor regarded by them as causative. The element most persistently spoken of is trauma. This is largely because of its medicolegal importance. The reports are not all of equal value, by any means, and the sequitur of cause and effect is not always convincing in the evidence given in the histories.

Schittenhelm [28] ranges himself on the side of those who believe that there is no true relation between tabes and trauma. His conclusions are based on a careful study of 128 cases of tabes in which he has made a searching analysis of all of the possible etiological factors. Sixty-four of his patients were frankly with syphilitic infection, and in 19 the infection was probable, but in at least 45 no history of infection could be obtained. Analyzing most of the reported cases called traumatic, Schittenhelm restricts himself to a discussion of 7 in which trauma was the only known pretabetic accident, but in none of these does he find sufficient evidence to prove the case.

On the other hand, we find a number of reports of tabes said to follow trauma. Risenfeld,[29] in a study on traumatic syringomyelia and tabes, gives the history of a man falling from a height upon the buttock. There was paralysis of the bladder following and anæsthesia in that viscus, with rapid development of pain and girdle-sense on the side of the lesion. Tabetic symptoms developed. Notwithstanding a positive denial of syphilis, a lumbar puncture revealed a lymphocytosis. The author reports it as an illustration of the hastening of the onset of a latent tabetic process, rather than as affording definite evidence of the relation of trauma to tabes. Brush and Hamilton, however, report histories which convey the impression that trauma may be an exciting cause. The former [30]

[28] Deutsche Zeitschrift f. Nervenheilkunde, xxiv, p. 432.

[29] Volkmann's klin. Vorträge, 380.

[30] Journal A. M. Assoc., 1904, xlii, p. 870.

gives the histories of 7 patients in whom he was unable to find a syphilitic lesion or information leading to the probability of syphilitic infection. No lumbar punctures are reported. Brush cites the histories in support of the idea, without himself advocating it, quoting, however, Golebiewski's view with reference to the same subject.

Pathology

EXPERIMENTAL CEREBROSPINAL MENINGITIS *

BY SIMON FLEXNER, M.D.

Of New York

Cerebrospinal meningitis has prevailed during the past two years, or is prevailing at present, as an epidemic disease, in the northeastern United States, northern Germany, and portions of Scotland. In the United States the chief focus of the disease has been in the vicinity of greater New York City, and in Germany the chief focus has been in Silesia. Recent reports indicate that the epidemic in Scotland prevails in the cities of Glasgow and Edinburgh, but whether more generally I do not know. During the winter of 1904 and the spring of 1905 about 4,000 cases of epidemic meningitis were reported in Greater New York City, of which 3,429 terminated fatally. In the year 1904 the number of deaths reported as due to epidemic meningitis was 1,403. In the year 1905, the number of cases of the disease reported was 2,755 and the number of deaths 2,026. The percentage mortality, deduced from the figures for 1905, is 73.5 per cent.

The high mortality of epidemic meningitis and the deplorable deformities caused by it demand that incessant efforts be made to discover therapeutic measures which may mitigate the consequences of the disease. A possible basis for improved therapeutics should be a better understanding of the pathology of the disease, and the production by experiments of the disease in lower animals which may be subjected to tests with therapeutic agents.

The epidemics through which New York and Silesia have recently passed have been scientifically fruitful in establishing more firmly the belief in the spread of the disease through immediate or

* Abstracted from papers which appear in The Journal of Experimental Medicine, 1907, vol. ix, pp. 105-187.

mediate contact with the sick, and in tracing the common occurrence of the causative germ in the nasal and pharyngeal secretions of the sick, and the exceptional occurrence of the microörganism in these secretions in the well who have been in contact with the sick. This mode of spread of the disease through directly and indirectly infected persons must come to exercise an important influence on the measures enforced hereafter to limit the spread of the disease.

The cause of the epidemic in New York and in Silesia is the Diplococcus intracellularis of Weichselbaum. This microörganism has been obtained in pure culture from many hundred cases of the disease, either during life by spinal puncture, or after death at autopsy. In exceptional instances it has been associated with other bacteria which, it is believed, entered the inflamed membranes secondarily. I have studied many strains of the diplococcus obtained by spinal puncture or at autopsy. The diplococci fulfilled the ordinary cultural requirements, and they showed the low degree of viability which has been observed commonly. I quickly learned to lay some stress upon the rapidity with which involution occurred in cultures which grew luxuriantly, and, later, I learned to trust the fermentative reactions of the diplococcus. Dextrose and maltrose are feebly attacked by the diplococcus, and acid is produced. Gas is never liberated, and in a sheep-serum-water-litmus medium, containing the sugars mentioned, reddening but no coagulation is produced. Certain other diplococci, of Gram-negative staining reaction also, with which Diplococcus intracellularis might possibly be confused, were either devoid of power to ferment sugars, or they possessed wider or more vigorous fermentative power than the intracellularis.

As stated, the brief vitality of many cultures of the intracellularis is a point of differential importance. Many strains, grown on a favorable medium, unless transplanted to a fresh medium, do not survive beyond two or three days. Indeed, these transplantations do not succeed, frequently, unless considerable quantities of the growth are carried to the fresh relief, an indication that only a fraction of the cocci have survived, or are still capable of multiplication. Vigorous fresh cultures of sixteen to twenty-four hours' growth stain sharply and uniformly. On the second day, the cocci show irregularities in staining and size, which irregularities are increased by desiccation of the medium and by other injurious causes. Cul-

tures three days old show marked degenerations, and these increase rapidly with age until, at the end of five or six days, or even earlier, no normal cocci persist. Even with very careful preservation, the great majority of the cocci do not survive beyond this period. A few cocci, in carefully preserved cultures of certain strains, may survive for many months. The appearance of old cultures is characteristic. At the beginning of the degeneration, swollen, deeply-stained cocci occur among the smaller but usual forms, which take the stain less intensely than fresh cultures. At this stage, tetradic groups appear, especially among the swollen cocci. As degeneration progresses, loss of staining and disintegration ensue, until, finally, staining power is entirely lost and a formless detritus remains.

Many experiments were carried out to ascertain the conditions affecting the brief viability of the diplococcus. From these tests certain conclusions can be drawn. In the course of growth of the diplococcus, the nutritive medium is not exhausted of all nutritive value for the coccus in twenty-four to forty-eight hours; and the products of disintegration of the diplococcus are inhibitive to further multiplication of the coccus only when they have accumulated in large quantities. It would indeed appear as if such an accumulation of growth products as to prevent further development could probably not occur in the course of ordinary vegetative existence of the organism; and it is safe to infer that the short vitality of the coccus in cultures is not due directly to the injurious action of these substances. The tests further bring out the fact that a fresh and vigorous culture upon a slanting solid surface succumbs quickly when kept constantly at a temperature just above zero, and is less injured by the wide variation of temperatures between 2° C. and 37° C. It could, therefore, be shown that cold is inherently injurious to the cocci, and the injury is not inflicted as soon as the low temperature is reached, or equally upon the cocci exposed to it. In view of the ease and certainty with which solid cultures are destroyed by cold, the greater resistance displayed by highly concentrated suspensions of the cocci in salt solution exposed to cold is noteworthy. Salt solution, even when charged with washings from the surface of a solid medium, probably does not permit any multiplication of the diplococci, besides which any increase was excluded by the low temperature. The factor of concentration of suspension

of cocci proved very important in respect to the preservation of cocci under the conditions of higher temperature when enzymotic action came actively into play. If a fresh culture, on a favorable medium, is suspended in salt solution to a milky mixture, and the latter is kept at 37° C., the greater number of the cocci introduced will be so degenerated at the end of twelve to fourteen hours as to be almost unrecognizable. As the suspension is diluted progressively, the degree of degeneration and disintegration diminishes. At the lower temperature (2° C.) more diplococci survive in the concentrated than in the weaker suspensions, while at the higher temperature, more of them survive in the weaker than in the higher concentration. A glance at the microscopical preparation gives a clue to this difference of behavior: while at the refrigerator temperature the diplococci are not markedly disintegrated, at the thermostat temperature, the degree of disintegration and concentration go hand in hand. Hence, while the diplococci may lose capacity to grow without losing, at the same time, their form and staining reactions, it is probable that once these latter are lost, viability is also lost. Cold, like salt solution, is inherently hurtful to the diplococcus, but, besides the injury which the salt solution alone inflicts, another detrimental factor comes into action at the thermostat temperature.

The nature of this detrimental agency is rendered fairly evident from the microscopical appearances presented by the suspensions, it would seem to be enzymotic. The manner of the dissolution of the diplococci suggests this, and the enzyme is doubtless contained in the bacterial cells. As enzymes act with energy not upon highly vitalized but upon dead or partly devitalized cells, the question arises whether in the case of the diplococcus the enzyme can act immediately as a devitalizing as it can as a dissolving agency. It has been shown that in weak salt suspensions, the diplococci survive and retain their morphological integrity longer than in strong suspensions; and hence, it may be conceived that while the charges of enzyme which individual diplococci carry may be inadequate both to destroy and to disintegrate them, that in the more concentrated suspensions the enzyme liberated from a larger number of disintegrating microörganisms, may exert a devitalizing effect. The intracellular enzyme is, therefore, a dissolvent of dead diplococci,

and in certain states of concentration a poison for living ones. The action of the enzyme upon the bacterial cells can be accelerated by the employment of chemical and physical agents—toluol, heat carefully applied—which serve to kill the diplococci without, at the same time, seriously injuring the enzyme.

A simple experiment has shown that the addition of a salt of calcium to an otherwise suitable culture medium will greatly increase the period of viability of the diplococcus which has grown upon it. If calcium carbonate is added to sheep-serum water agar a given culture will survive many weeks. Ringer's fluid—*i.e.,* salt solution containing small amounts of calcium and potassium chloride —preserves the diplococci from rapid death and disintegration. Salt solution is directly injurious to the diplococci, and the toxic effects of sodium chloride can be neutralized by certain calcium and potassium salts. By increasing the calcium content of a culture medium the viability of the diplococcus can also be prolonged.

Diplococcus intracellularis is, as tested on laboratory animals, a microörganism of low and variable pathogenic action. Not all cultures of the diplococcus, even in the first generations, are even moderately pathogenic for guinea pigs and mice, although it is always possible to kill these animals by inoculating an excessively large quantity of cultures of low virulence. Speaking generally, the freshly isolated cultures are much more virulent than cultures grown for a period on artificial media. Occasionally, a strain of the diplococcus will retain its virulence for many months, but, as a rule, this power becomes greatly diminished, or is lost, after the first transplantations extending over a few weeks.

The fate of the diplococci injected into the peritoneal cavity in guinea pigs depends upon several conditions: the quantity of diplococci injected, the virulence of the diplococci, somewhat upon the individual resistance of the pigs, since this is a factor in prolonging or diminishing the period of survival, and the extraneous condition of elapsed time between death and autopsy. The more virulent the diplococcus the shorter is the period of survival after inoculation. Guinea pigs which succumb in less than eighteen hours tend to develop a peritoneal exudate poor in leucocytes; while the pigs which survive longer, *i.e.,* from twenty-four to thirty-six hours, tend to exhibit turbid exudates rich in leucocytes. With an increasing

period of survival there is greater emigration of leucocytes and also greater disintegration of diplococci. Hence, the degree of emigration of leucocytes comes to be a measure, in part, of the degree of virulence of the diplococci. It should be pointed out that large overdoses of the weaker diplococci can be readily disintegrated in the peritoneal cavity, even though emigration of leucocytes is largely prevented by the excessive dose. From this fact the result may be deduced that the destruction of the diplococci does not take place necessarily within leucocytes, but can be accomplished by fluid inflammatory exudates alone. There is similarity in the pathological conditions met with in cerebrospinal meningitis in man and in the experimental infection in guinea pigs. In both species the immediate local reaction to the presence of the diplococcus in the serous cavities is an exudation of fluid, the character and quantity of which varies greatly, perhaps with the quality of the infecting diplococcus. The less cellular fluids often contain the great number of diplococci of well-preserved appearance which are readily cultivated; while the more cellular fluids often show fewer diplococci and yield cultures with greater difficulty.

Since in cerebrospinal meningitis in man, cultivation experiments may fail to yield the diplococcus, although the microscope shows it to be present, attention may be called to similar failures with guinea pigs. In the case of the guinea pigs those succumbing earliest give the greater number of successful cultivations. But if the autopsies are delayed and partial decomposition of the body has already set in, or if the dead pigs have been kept for many hours in the refrigerator, the chances of successful cultivations of the diplococcus are greatly reduced.

In view of the manner in which Diplococcus intracellularis acts in causing death of guinea pigs, it may be assumed that its effects are produced by a poison liberated from the bacterial bodies, probably through disintegration, and not to a secreted extracellular poison. Multiplication on the part of the diplococci is not at all essential to the production of the peculiar symptoms and lesions. In conformity with this view is the fact that cultures killed by heat and the autolyzed fluids are highly toxic.

At the beginning of this study I had the plan of using monkeys to reproduce the symptoms and lesions of cerebrospinal meningitis

in man. Some of my earliest experiments were made with monkeys. The cultures were introduced into the spinal canal by means of lumbar puncture. I have found that the injection of relatively large quantities of living diplococci into the muscles about the spinal column, or into the muscles and subcutaneous tissues of other parts of the body, produces only trifling effects. Not a few cultures have failed to produce symptoms or lesions when injected into the spinal canal of monkeys. Virulence plays an important part in these injections.

The monkey offered itself as a suitable animal for the inoculation experiments because of its relationship to man; the fact of its upright carriage, which would permit comparison with man in respect to the distribution of the lesions, and because of the ease and safety with which repeated lumbar puncture could be made in following the course of the disease.

It was shown by the experiments that in periods of from fourteen hours to two or three days advanced and deep lesions can be produced in the brain and its membranes by Diplococcus intracellularis. The inflammatory exudates resemble in character those present in acute cases of epidemic meningitis in human subjects. The distribution of the exudate chiefly over the spinal cord and basal ganglion bears comparison with the conditions found commonly in man; and the involvement of the spinal nerves and ganglia, the nasal mucous membrane, and the arteries in the inflammation is especially noteworthy. It is usual to find in the monkeys succumbing to the experimental infections, evidence of inflammation of the nasal mucosa. Smear preparations from the superior nasal mucosa often show variable numbers of leucocytes enclosing diplococci presenting the morphological and staining properties and the degenerations of the diplococci in the brain and cord. If the dura mater beginning at the olfactory bulbs, surrounding the olfactory nerves, and extending through the cribriform plate into the nose, be carefully removed with the adjacent portion of the ethmoid bone and olfactory mucous membrane, fixed in Zenker's fluid, sectioned longitudinally, stained in hæmatoxylin or methylene-blue and eosin, and examined microscopically, the passage of leucocytes from the brain cavity into this membrane and about the olfactory nerves towards the ethmoid, can be traced. The blood vessels of the adjacent olfactory mucous mem-

brane are seen to be dilated and leucocytes to be passing into the layer of columnar epithelium of the surface. The abundant lymphatics of the mucous membrane are in communication with the lymphatic spaces which enclose the branches of the olfactory nerves, and these spaces again communicate with the subdural and subarachnoid spaces of the cranium, so that the lymphatics of the nasal mucous membrane can be injected, according to Schwalbe and Key and Retzine, from the cranial cavity.

I have not succeeded, although I have made several attempts, in cultivating Diplococcus intracellularis from the nasal mucosa of these monkeys. I realize, of course, that it is highly desirable and very important to bring this final proof of the passage of the diplococci from the cranial cavity into the nose. The difficulties which surround the demonstration are considerable, for I think it probable that the diplococci do not long remain viable in the nose in monkeys. I conceive the conditions there to be, possibly, even more unfavorable to them than in the meninges where, indeed, they tend rather to disintegrate than to multiply. The conditions in man are, of course, very different and far more favorable to the existence of the diplococci. But in spite of the incompleteness of the proof in the case of the monkeys, I would still urge that attention be given to this possible mode of infection of the nasal and pharyngeal passages in human subjects.

The experiments indicated that the injected diplococci tend, regularly, to be distributed upwards along the spinal cord and the base of the brain. The invasion of the pia of the convex and mesial surface of the brain takes place at a later period, and the infection of the ventricles also takes place later, either because the diplococci are delayed in reaching them, or because they resist infection longer. There is, moreover, relation between localization of the diplococci and the degree and extent of the exudation. Where the accumulation of inflammatory products is most marked the diplococci will, if still present at all, be most abundant. Finally, the growth of the diplococcus upon a suitable culture medium will be greatly influenced, if not wholly determined, by the conditions to which the infected body is subjected after death. While a temperature just above the freezing point will preserve the morphological elements of the body and even the body of the diplococcus, yet if it is

maintained for many hours, it may render the diplococcus incapable of multiplication. Higher temperatures subject the body not only to autolytic and putrefactive processes which interfere with the pathological examination, but they deprive even more quickly than cold the diplococcus of power of growth and, under certain conditions, of staining properties as well. The temperature at which the body is kept, and the elapsed period of time between death and the examination, have a direct bearing upon the cultivation of Diplococcus intracellularis from the infected body. I am of the opinion that these factors operate in much the same way in cases of human meningitis as in the case of the artificially infected monkeys and guinea pigs. Certain discrepancies in the bacterialogical studies of cases of meningitis in man may, in view of these considerations, receive an explanation.

To insure a fatal outcome a diplococcus of known virulence in a sufficient dose must be injected into the spinal canal. Individual differences in susceptibility occurred among the monkeys, but no monkey was wholly refractory to inoculation. As compared with the doses which probably determine infection in man those used to produce the experimental disease in monkeys are colossal.

In contrast to the ease with which the acute inflammations can be excited is the difficulty which attends the production in monkeys of a subacute form of meningitis. The intraspinal injections produce either an acutely fatal meningitis, or an acute disease from which recovery takes place rapidly. The animals which survive inoculation are usually well of the disease in three or four days. In rare instances, the animal lingers a victim of the disease for a longer period than this to succumb in the end. Monkeys which survive the second day after inoculation tend rather to recover than to die. By following in the inoculated monkeys the changes in the cerebrospinal canal by means of lumbar puncture, the progress of the infection can be traced and the result often predicted. Disappearance of the diplococci early from the canal is a good sign; early emigration of leucocytes is also a good sign; early emigration, active phagocytosis and dissolution of the diplococci, both within and without leucocytes, are very favorable signs. Proceeding in this way, I have been able to keep a small number of monkeys in a state of subinfection with the diplococcus for a period of several

days or weeks. The period of successive inoculation was determined by the puncture and by the physical condition of the monkey. A state of resistance tended to develop which necessitated the employment of increasing amounts of cultures to produce visible effects; and in the end the run-down animal succumbed to the dose administered. In these animals the exudates were thicker and firmer and covered the base and the convex surface of the brain, and the ventricles were sometimes widely dilated and contained turbid exudates.

By means of the experiments upon which the foregoing views are based, it has been shown that the lower monkeys can be infected without great difficulty with Diplococcus intracellularis and made to reproduce the pathological conditions and, to a certain extent, the symptoms present in man in cerebrospinal meningitis. In comparing the experimental lesions in monkeys with the naturally developed lesions in man, note should be taken of the occurrence under both conditions of encephalitis and abscesses, of hæmorrhages, of proliferation of large cells of connective tissue and tissue spaces, of acute endarteritis, of inflammation of the dorsal root ganglia, of internal hydrocephalus, of relatively small amount of fibrin in the exudates, of fibrinous and other thrombi, and of phagocytosis of diplococci and somatic cells.

The experiments, some of the results of which have been set down here, besides being an argument for the causative action of Diplococcus intracellularis in epidemic meningitis, form, also, the basis of an attempt to influence the progress and termination of the experimental infection in monkeys through the employment of antisera prepared from the diplococcus.

The one therapeutic measure growing out of the study of the recent American and German epidemics of meningitis which offers any hope to the world is an antiserum for the diplococcus. It is true that the experience of the past is not favorable to the hope of achieving remarkable success by the employment of antibacterial immune sera. All indications point to the pathological effects of the diplococcus as being caused by endotoxic constituents; and thus far, according to many investigations, these endotoxins have failed to yield, by methods of immunization, active antisera which have proved valuable in the treatment of infective diseases. Opinion is,

however, considerably divided on this subject; and in the absence of more certain methods tests of antisera for the diplococcus are certainly justified. These tests can in the preliminary stages be carried out on certain animals, since the course of infection in them with the diplococcus is now fairly well understood.

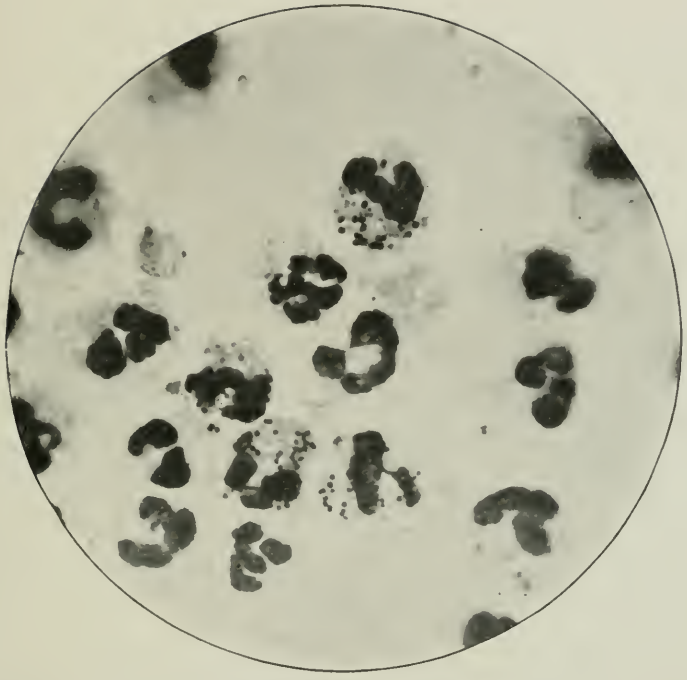
The main question which would seem to be involved in the search for an active antiserum against meningitis is whether the quantity of antibody which can be produced will suffice to neutralize such a quantity of the poison of the diplococcus as to influence the result of the infection. In fact, the problem may not be so simple, or, indeed, so hopeless as this proposition indicates. It is, of course, important that neutralization of the poison should if possible be secured, but the effects of restraint of growth and multiplication of the diplococcus may, at some periods of the disease, be of greater significance than the neutralization of free endotoxin. Fortunately many agents, some of them quite indifferent, are able to affect the power of multiplication in the body of the diplococcus. It has been shown, indeed, that serum in the fresh state and after heating to 60° C. preserves the power to destroy in test-tubes large numbers of the diplococcus, and sterile fluid inflammatory exudates possess this power in even greater degree. An antiserum, therefore, even though it contains relatively small amounts of antibodies, as indicated by neutralization experiments, may be effective beyond this calculated value by restraining the multiplication of the diplococci, possibly by reducing outright their number, and by supporting the power of resistance normally at the disposal of the body.

The conditions are made theoretically less discouraging, perhaps, because the main pathological lesions are limited to the cavity of the cerebrospinal axis. A large advantage is gained by this circumstance. If we are at all permitted to apply test-tube experiments to what may happen in the body, it would not be remarkable if the normal serum of animals, and perhaps of human beings, proved to be beneficial to a degree when brought into direct relation with the focus of development of the diplococcus. At first sight, judging from test-tube experiments, it would appear as if the exudate called out by the inflammation, should suffice to destroy the diplococci; this manifestly does not happen in many cases. Indeed, it is found that incubation outside the body will even increase the number of

diplococci in the inflammatory fluid withdrawn from the spinal canal. It is safe to assume, therefore, that the exudate withdrawn has been exhausted of its power to destroy the diplococcus. It is quite possible that the introduction of fresh serum, of the same species of animal, may be helpful by bringing quickly into contact with the diplococci a quantity of actively destructive serum. The results of some of my experiments show that normal serum reduces appreciably the toxic effect of given doses of the diplococcus.

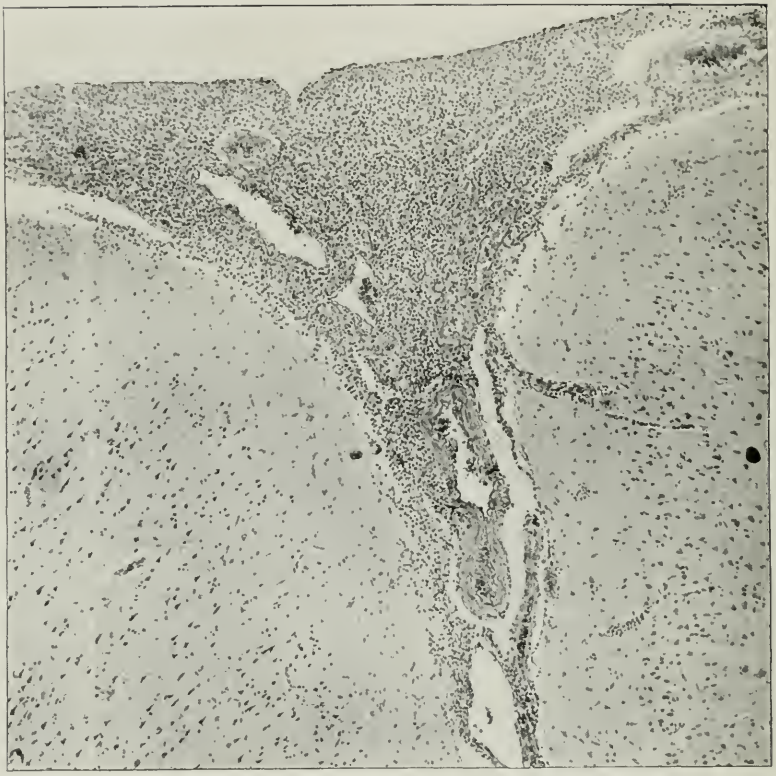
I am far from having any conviction that cerebrospinal meningitis in man can be influenced favorably by injections of immune sera into the spinal canal, or elsewhere in the body. My experiments merely show that guinea pigs and monkeys, in which the conditions of infection can be controlled, can be saved from the otherwise fatal effects of the diplococcus by the use of antisera, and to a less extent by the use of normal sera and other fluids (bouillon). The experimental results with the antisera were not sufficiently constant and striking to make this mode of treatment of meningitis of very hopeful augury. On the other hand, it is not improbable that more active antisera, using appropriate means of immunization, may be produced. Possibly, such antisera may prove of value in the treatment by direct spinal inoculation, possibly even by intravenous or subcutaneous injection, in this hopeless disease. The evident disadvantages to which the human patient must always be subject, as compared with the animals used for experiment, arise from the difficulty often encountered of estimating exactly the duration of the disease, and applying the remedy at the most favorable stage. On the other hand, the exceptional cases only in man run so rapid a course, attended with such profound symptoms of intoxication, as are regularly seen in the inoculation disease in animals. The slower and more measured progression of the infection in human beings may, indeed, be a favorable circumstance, provided the treatment can be applied in the early stages and before too severe structural changes have taken place in the nervous system. The fact that normal serum exercises a certain degree of protection might possibly be taken advantage of in cases of human infection. It would, of course, be practicable to obtain normal human serum for such injections. The subject is one which, in view of the gravity of cerebrospinal meningitis in man and the absence of any efficient therapeutic measure against it, would seem to deserve consideration.

Fig. 1.



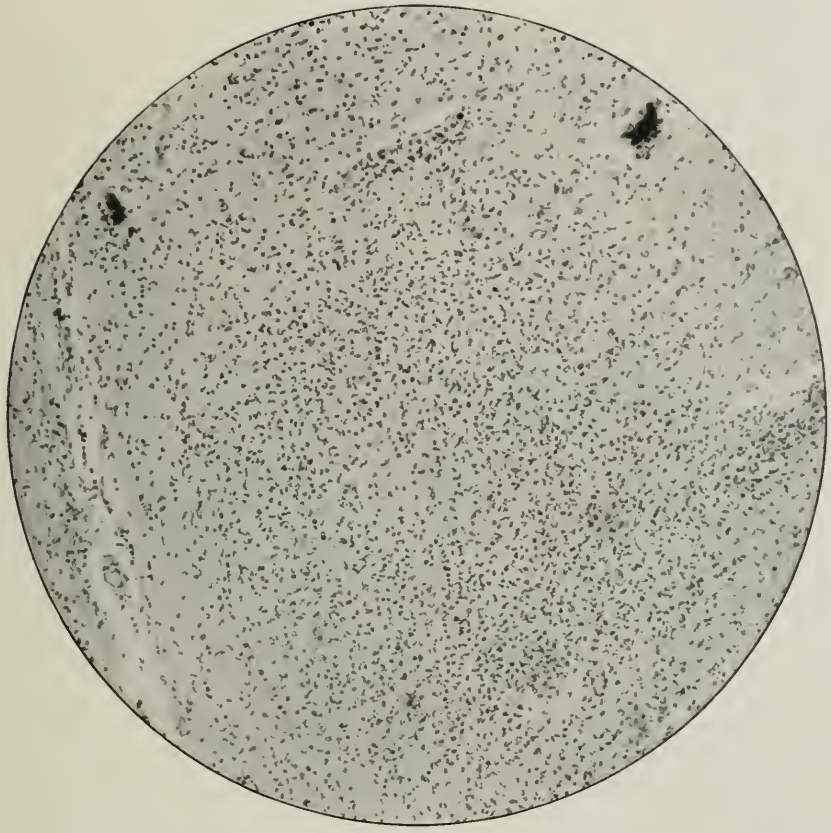
Smear preparation from contents of brain abscess, monkey No. 1. Typical intracellular diplococci. Methylene-blue staining. One-tenth inch objective.

Fig. 2.



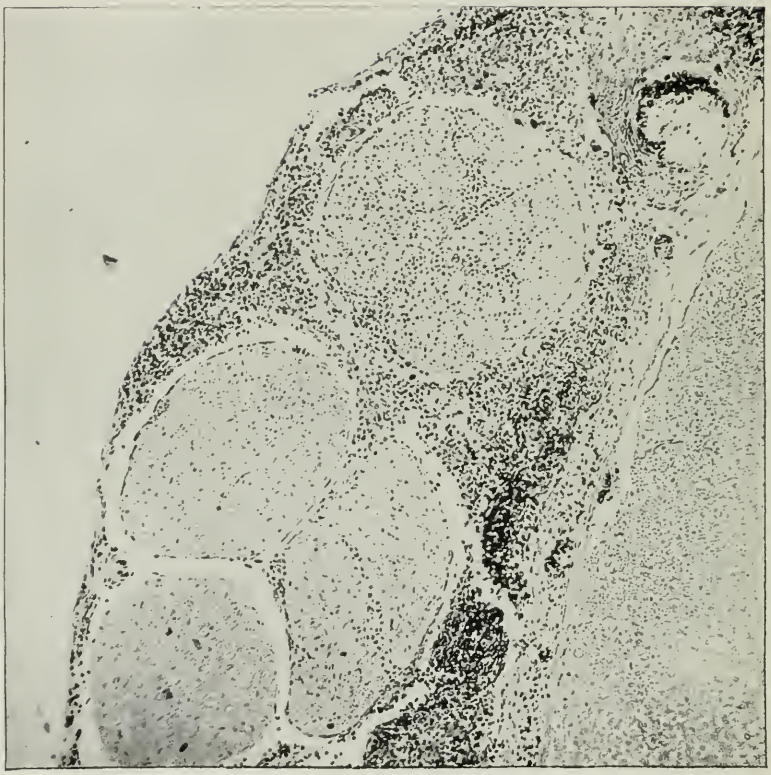
Low magnification to show the relation of the exudation in the pia-arachnoid of the convex surface of the brain and the sulci, and the extension of the exudation along the cortical vessels into the brain.

Fig. 3.



Low magnification to show the degree of acute encephalitis, and the involvement of intracticalor blood vessels in the infectious processes.

Fig. 4.



Low magnification to show the exudation about and within the spinal nerves.

THE PATHOLOGICAL ANATOMY OF GENERAL INFECTION WITH THE GONOCOCCUS

BY JOSEPH EDGAR TYREE

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Thayer and Blumer[96] in 1895 for the first time cultivated the gonococcus of Neisser from the blood of a patient who subsequently died, and upon whom an autopsy showed the cause of death to be a gonococcal endocarditis and septicæmia. Lenhartz[66] in 1897 inoculated a puriform mass which contained gonococci, from the heart valves of a patient dead of an endocarditis, into the urethra of an already fatally ill man; a typical gonorrhœal urethritis developed.

Since these experiments, many articles have appeared on the subjects of gonococcal endocarditis, general gonococcal infection, pyæmia and septicæmia. Most of the authors have realized that the gonococcus is capable of causing the lesions common to the pyogenic organisms, and the opinion is growing that this coccus frequently finds its way into the blood stream and does other harm than that caused by its growth in the urogenital organs. But, as yet, the cases with autopsy reports have not been collected to such an extent that a general picture of the pathological anatomy of gonococcal septicæmia can be formed. This fact became evident in the study of a typical case,[100] the autopsy report of which is given below. It is the aim of this paper to place general infection with this organism in the class with streptococcus septicæmia, typhoid fever, pneumonia, scarlet fever, diphtheria, etc. The term gonococcal is used in preference to gonorrhœal, since it expresses the true condition—merely an infection with a specific organism.

HISTORY OF THE WRITER'S CASE

The subject was an unmarried woman, twenty-two years of age, a nurse by occupation, and a patient in the Englewood Union Hospital in the service of Dr. J. H. Hess, to whom I am indebted for the history of the case. The chief points of interest are the follow-

ing: The patient had been quite healthy until three years previously, when she was operated on for appendicitis; a year later there was an operation for post-operative adhesions, and about the same time an illness diagnosed as typhoid fever. One year ago there occurred an attack of rheumatism in the left fore-arm. Four months previous to her entrance to the hospital the patient had an attack of specific suppurative ophthalmia, from which she recovered completely. Early in August, 1905, she began to feel unwell, complaining of occasional chills and some fever. During menstruation she was exposed to cold, following which the flow ceased at once, and three days later she developed a left-sided pleurisy. Two days later an attack of rheumatism began, with swelling of several joints, but especially the right ankle and knee and both wrists. A few days later, August 30, she entered the hospital, with high temperature, pulse and respiration. At first she was delirious. Rheumatic pains and swelling developed in most of the joints, finally localizing in the right knee, which became partially ankylosed. On the third day after entering the hospital hæmorrhagic vesicles appeared over both elbows and ankles, with a few scattered over the buttocks and abdomen. These persisted for some time, but gradually disappeared, leaving deep, pigmented scars. Loud cardiac murmurs developed, indicating both stenosis and incompetence of the aortic orifice. A leucocytosis of from 20,000 to 29,000 was present throughout the disease. Death came from exhaustion on October 22, and autopsy was performed a few hours after death by Dr. H. G. Wells.

AUTOPSY REPORT OF CASE

Anatomical Diagnosis.—Acute vegetative and ulcerative aortic endocarditis. Acute myocarditis. Slight hypertrophy of left ventricle. Anæmic infarct in spleen. Subacute splenic swelling. Acute parenchymatous nephritis (large, yellow kidney). Absence of appendix vermiformis. Healed laparotomy wounds with adhering omentum. Bilateral fibrous pleuritis (slight). Contracture of right knee joint. Subcutaneous ecchymoses. Acute endometritis. Fibrous perioöphoritis. Thrombosis of central vessels of left adrenal, with acute inflammation of the adrenal.

External Appearances.—The body is that of an emaciated

young woman. Height, 122 cm. Scleræ are white and conjunctivæ show no change at present. Superficial lymph glands are not palpable. There are two parallel scars over McBurney's point. Right knee lies flexed at about 40 degrees; cannot be entirely straightened, but permits flexion to right angle. There is a purplish subcutaneous discoloration over inner side of right heel, and a small decubitus over the right internal malleolus. There are a few superficial circular scars about 2 mm. in diameter over various parts of the body. Below the clavicles is an erythematous reddening, disappearing on pressure. Over the abdomen and under the left breast are innumerable small, punctate red spots, not disappearing on pressure. The second and fourth ribs are unusually prominent. There is a small ulcer over the sacrum.

Abdominal Cavity.—On opening the abdomen the omentum is found nearly free from fat, and adherent to the parietal peritoneum beneath the superficial scars; these adhesions also join the colon and lower end of the ileum together and bind them to the parietal wall. At one place the tip of the finger can be pushed through the rectus muscle to the skin, at the bottom of the first scar. Both inguinal and femoral canals are closed. The margin of the liver is above the costal arch. The appendix is entirely missing. Numerous fibrous tags, often pigmented, are scattered over the ileum; portions of the ileum are much congested. Mesenteric glands are not enlarged or reddened. Peritoneum in general is smooth and shining, and the cavity is free from fluid. There are a few soft, fibrous adhesions about the ovaries. The uterus is normal in position, but a little enlarged. Lower pole of both kidneys reaches to opposite the body of the fourth lumbar vertebra. The lesser omental cavity shows no changes. A few adhesions about the spleen. No changes seen in the pancreas. No adhesions about the gall bladder.

Thoracic Cavity.—There is no thickening of the costo-chondral junctions. On opening the chest the pericardium is seen to be exposed over a large surface. Left lung shows many soft, fibrous adhesions to the pericardium and diaphragm; no fluid in left pleural cavity. Right lung has a few adhesions about the apex. Thymus gland entirely replaced by fat. No changes in anterior mediastinum. On opening the pericardium a few ecchymoses are seen on its parietal surface; otherwise it is unchanged.

Heart.—Left ventricle somewhat enlarged. Aortic valves competent to water test. Pulmonic valves appear normal. Mitral valve slightly thickened; smooth and quite normal from both sides. Myocardium is pale. Blood has a watery appearance. No changes in the auricles. The aortic valve is covered with large, yellow, fibrinous excrescences, which nearly occlude the orifice. The chief mass —about 10 x 12 mm.—is on the ventricular surface of the right posterior cusp, although smaller masses are present on both the other cusps and also on the aortic surfaces of the cusps; on the right posterior cusp is an area about 2 mm. in diameter, which has ulcerated through the valve, but the opening is partly occluded by the thrombotic mass. At the base of the right posterior cusp on the ventricular surface is a small mass bulging beneath the endothelium, and consisting of a valvular aneurism filled with a soft clot which is continuous with the vegetation behind the valves. The valves are not at all thickened or sclerotic. The wall of the left ventricle is slightly thickened, being 12 mm. thick. There is no sclerosis in the aorta.

Lungs.—The left lung collapses and crepitates throughout; is slightly congested posteriosly; nothing abnormal palpable. Contains a little frothy fluid. The right lung is like the left. Both show very little anthracosis.

Spleen.—Much enlarged, but symmetrically. Weight, 500 grams. Measurements, 18 cm. x 10 cm. x 6.5 cm. On the surface at one point is a light yellowish area, 1 cm. in diameter, surrounded by a congested zone. The yellow area extends 15 cm. into the organ and is sharply circumscribed and surrounded by an area of congestion. Consistence about normal. The splenic substance is firm and reddened in places. Malpighian bodies not easily seen. The increase in the size of the spleen seems due to increase in the pulp.

Liver.—Does not seem to be enlarged. At the hilum is an enlarged, firm lymph gland. The liver substance is rather pale; otherwise no change. The gall bladder is distended with a thick, tarry bile, and contains no concretions.

Pancreas.—Appears to be normal.

Adrenals.—One, the left, is quite firm and redder than normal.

Kidneys.—Left: Greatly enlarged and firm. Measures 15 x 6.5

x 5.5 cm., and weighs 340 grams. It is about normal in consistence. The cut surface is yellow, slightly mottled with red and pink. The blood vessels are congested. Glomeruli glistening, some red and some pale. Cortex measures from 10 to 12 mm. in thickness. Pyramids are 20 mm. thick. There are many large submucous ecchymoses in the renal pelvis. The capsule strips very easily. Right: Is slightly smaller than the left, measuring 12 x 6 x 5 cm. In all other respects it answers to the description of its mate.

Ovaries.—Normal in consistence. Contain healed and healing corpora lutea. A few fibrous adhesions are found around them.

Uterus.—Slightly larger than normal, softened and reddish. Cervix contains a plug of mucus, and more mucus can be expressed from the uterine cavity. Endometrium covered with this mucus and somewhat ecchymotic.

Fallopian Tubes.—Show no changes.

Intestines.—Mucosa of lower end of ileum covered with mucus and somewhat congested. No other changes visible. No external abnormalities in the intestine.

HISTOLOGICAL EXAMINATION

Kidney.—There is quite a marked congestion, with a large proportion of leucocytes in the blood. The convoluted tubules are filled with leucocytic masses, consisting mainly of polymorphonuclears with a few lymphocytes. There are also many of these same cells in the loops of the glomeruli, but very few erythrocytes. In many of the Malpighian corpuscles there is a proliferation of the cells of Bowman's capsule, forming the so-called "crescents." There is very little parenchymatous change, and practically no interstitial change. The straight tubules are normal and contain no abnormal constituents. Sections stained by sudan III show fat granules in but a few of the convoluted tubules and none elsewhere. Total amount of fat extremely small.

Liver.—The liver shows an infiltration of various kinds of leucocytes—principally polymorphonuclears—in the Glissonian tissue at the periphery of the lobules. There is a slight central fatty degeneration, but no other changes.

Spleen.—This organ shows great active congestion, and a great many polymorphonuclear leucocytes and large phagocytes in the

splenic pulp. The infarct described in the gross specimen shows beginning encapulation.

Adrenal (left)—The adrenal shows a marked congestion in the cortex, with even more marked leucocytic infiltration than seen in the kidney. There are also collections of polymorphonuclear leucocytes in small patches in the medulla. Granular parietal thrombi occupy all the large vessels in the central portion of the organ, leaving only very small lumina in the vessels, which are filled with a recent fibrinous clot.

Myocardium.—Shows small patches filled with polymorphonuclear leucocytes, but without proliferation or degenerative changes. This infiltration is especially marked just beneath the endocardium and around the blood vessels.

Uterus.—The mucosa is congested and shows a slight leucocytic infiltration. The surface epithelium is lacking. The glands are nearly normal.

Fallopian Tubes.—Seem perfectly normal.

Ovary.—Vessels dilated. No Graafian follicles present in section. No acute inflammation.

BACTERIOLOGICAL REPORT

Smears from the vegetations showed them to consist chiefly of great numbers of diplococci, destained by Gram's method; they were of the so-called biscuit shape, with also many involution forms. Smears from the blood of the abdominal aorta, taken through the renal artery, showed a few of the diplococci free in the blood. Smears from the cut surface of the spleen showed the same organisms in the large phagocytic cells of the pulp, often in great numbers; but most of the cocci were in masses, and free. Smears from the cervix and body of the uterus showed the same cocci, along with many bacilli. On bouillon, glycerine agar and fresh hydrocele fluid, no growth was obtained except in one bouillon tube, which was contaminated.

This, then, is a somewhat generalized infection with an organism giving the morphological and tinctorial characteristics of the gonococcus of Neisser. The absence of growth on all culture media may be considered corroborative evidence that the only organism present was the gonococcus.

GENERAL CONSIDERATIONS

It has been possible to obtain definite data from some of the cases reported; but many others, even with autopsy reports, have contributed nothing to an accurate knowledge of other parts than the heart valves. This is, presumably, due to the fact that the examiners considered the conditions to be more like those found in endocarditis following acute articular rheumatism than those caused by septicæmia. The clinical picture of gonococcal sepsis is so similar to that of other systemic infections, however, that it seems but natural that we should find corresponding anatomical changes.

The tissue changes in local gonococcal infections, such as urethritis, epididymitis, conjunctivitis, vaginitis, etc., have been frequently described accurately in the literature, and hence will be included merely incidentally in this paper.

Etiology.—No case of cryptogenic gonococcal septicæmia has been reported; it always arises as a sequel to a demonstrable infection. It seems evident that the organisms are capable of entering the circulation from any of the local lesions. General infection resulting from gonorrheal conjunctivitis has been reported (Höck [51] and Brehmer [5]); but, of course, by far the most frequent antecedent is urethritis in the male, and the corresponding genital infection in the female. General infection may result without any further local lesions than those of the primary infection (von Leyden [73]); but, more or less often, epididymitis (Cordell [24]), prostatitis (Wasserman [107]), lymphadenitis (Columbini [22]), and many other local inflammations intervene. Both the blood and lymph streams may be the means of transporting the organisms from the original site to the general circulation. Hansteen [44] first positively showed this function of the lymphatics, finding gonococci in both the lymph sinuses and blood spaces of the inguinal glands of a patient suffering from urethritis. According to Rosenthal [88] the onset of the general symptoms is usually between the fourth and sixth weeks of the local infection, although the time elapsing may be long, as in one of Harris and Dabney's [46] cases; here the latent period was several months. When one considers the abundance of the blood supply, the plexiform nature of the veins (prostatic and

uterine), and the sinuses of the corpora urethræ and of the uterus, it would seem to be an easy matter for the gonococci to enter the blood stream from urogenital infections. Sepsis may occur at any time after the erosion of the mucosa and the entrance of the cocci into the deeper tissues; and since they may live here for an indefinite time, in para-urethral abscesses (Rana [82]), and in prostatic abscesses, the onset of general symptoms, even years after a urethritis, seems easily explained.

In defining what constitutes general infection with the gonococcus, we must consider the following conditions:

(1) When the organisms are found in the blood during life.

(2) When, at autopsy, the gonococci are found in the vegetations on the heart valves, in the spleen or other organs, or in the blood of the heart and vessels.

(3) When there is observed clinically a decided affection of the heart valves, preceded by the signs of an intoxication, which is subsequent to a gonorrhœal urethritis or conjunctivitis.

(4) Polyarticular rheumatism, in which the gonococci are found in the joints.

(5) Multiple, generalized abscesses containing the gonococci.

One and two are the only conditions in which we can positively make a diagnosis of gonococcal septicæmia. Cases illustrating 3 have sometimes given a mixed culture of gonococci and other pyogenic organisms (Harris and Dabney [46]; Hektoen [47]; Finger [36]); and sometimes only the complicating organisms (Fürth and Weber [38]). Also, as pointed out by McDonnell,[75] patients giving a history of recent scarlet fever and other infectious diseases in addition to gonorrhœa, cannot present a clear case of gonococcal endocarditis. As to 4 and 5, we may say that the organisms have wandered by the blood (Wertheim [106]), or lymph stream (Balzar [7]) from the original site, and, without appreciable multiplication in these fluids, have formed metastases in the tissues where they have lodged; we may call them conditions of pyæmia, realizing that septicæmia may result more easily when such conditions are present than when the infection is single or primary. His,[49] for example, cites a case of septic phlebitis of the pelvic veins from which general infection followed. However, such extensive suppurative conditions as those described by Crosby,[20] where the whole

respiratory tract, as well as the eyes, mouth, and urogenital system, were the seat of gonococcal suppuration, may not result in septicæmia.

PATHOLOGICAL ANATOMY

Heart.—Since the heart is the most constantly and severely affected of all the organs, and its condition is of the most importance to the body economy; and also, since it has been investigated most thoroughly in the disease under consideration, it seems well to describe the pathology of this organ first.

The heart valves are the chief point of attack, and hence we speak of the heart lesion, generally, as an endocarditis. According to Loeb,[70] those valves are commonly affected which are already the seat of some pathological process, or have at one time been damaged. Even when normal valves are attacked, those parts which are subject to the most wear and friction are the ones picked out. Hence it is found that the valves of the left side of the heart are far more frequently involved than those of the right, and of these, the aortic more often than the mitral. Lartigau,[65] in an analysis of sixteen cases, gives the valves affected as follows: Aortic, 8; mitral, 3; both, 2; tricuspid, 1; pulmonary, 2. Thayer and Lazear,[97] in thirty-one cases, found: Aortic, 12; mitral, 6; both, 3; pulmonary, 7; tricuspid, 3; mitral, aortic and tricuspid, 1; all four, 1. In fifty-four undoubted cases from the literature, I find the aortic valves affected in 28 cases, mitral in 16, tricuspid in 3 and pulmonary in 7.

Like acute endocarditis from other infective agents, that from gonococci may be ulcerative or vegetative, or, as in our case, a combination of both. In the fifty-four cases mentioned both processes were present in forty-two; the remaining twelve were vegetative only; no case was noted without at least slight vegetations.

The vegetations may be verrucose or warty, and form along the edge of the valve; they may be stringy, soft and friable, and hang as moss-like appendages in the blood streams; these last show areas of necrosis and have a great tendency to break off and form emboli. The vegetations may become calcified, as in a case reported by Loeb,[70] where there were hard calcified nodules on the posterior and anterior aortic segments, projecting into the ventricle. In some cases the vegetations consist of fresh coagula in the nature of

thrombi; His [49] reports such a case in which the excrescences were composed of fibrin and fresh coagula; microscopic examination showed fibrin masses with leucocytes in the free margin. In some instances the principal part of the growth seems to come from the injured tissue in the form of an exudate; our case is an example of this. It would seem that the growth, being firmly bound to the valve, would not be easily torn off; but the free parts become necrotic. Stein [94] reports a case where the vegetations on the mitral valve were 4 cm. long, and floating in the blood current.

On microscopic section, the polypoid growths are seen to consist mainly of fibrinous exudate with leucocytes, *i.e.*, white thrombi (von Leyden [73]); but these are probably only a stage in the formation of the more friable necrotic masses which may then go on to calcification, if the infection is mild and chronic enough. The vegetations on the several valves do not differ from each other in character.

The cocci are found constantly, in and on the excrescences, and in large numbers, when proper methods are used for their identification. They are found lying within the polymorphonuclear leucocytes, and also free in the coagulum or exudate; whether the extracellular cocci are degenerated or not has not been ascertained.

The most frequent site for the ulcerations is the base of the valves carrying the vegetations. Many varieties of these lesions have been described. In Loeb's [70] case the posterior and anterior aortic cusps were perforated. In the case of Bjelogolowsy [12] there was slight perforation of the aortic valve; Harris and Dabney,[46] report ulceration through the septum ventriculorum, and almost complete destruction of the aortic segments; Sears [91] reports a case with rupture of the aortic cusps; in Lartigau's [65] case the mitral cusps were destroyed by ragged perforations at points of attachment to the wall; in Winterberg's [108] case both aortic and pulmonary valves were completely destroyed; only fibrous, caseous tags remained. Aneurysmal dilatation of the base of the cusps was present in the cases of Michaelis,[76] Hektoen [47] and in our case. Frendel [39] reports aneurysm of the right sinus Valsalvæ and ulceration of the right aortic cusp.

The *myocardium* shows many grades of degenerative changes. In our case there were small areas between the muscle fibres and

under the pericardium and endocardium, filled with polymorphonuclear leucocytes, many of which contained gonococci; but there were no degenerative changes in the muscle fibres. In Frendel's [39] case, and that of Michaelis,[76] there was present a distinct cloudy swelling. Harris and Dabney [46] report œdema and fragmentation of the myocardium. Prochaska [80] found small collections of leucocytes under the endocardium and throughout the muscle; some contained the diplococci. His [49] found abnormally large nuclei in some of the muscle cells, such as are often found in the dilatated hearts in lobar pneumonia; collections of round cells were present between the muscle fibres. Sears [91] found infarcts in the left ventricle, Krause [62] reports fatty degeneration of the myocardium. In Councilman's [26] case, there was an infiltration of pus cells along the connective tissue, apparently extending from the endocardium; large areas of necrosis with purulent infiltration, extending from endocardium to pericardium, together with various degrees of degeneration of the muscle fibres, such as disappearance of the nuclei, swelling and vacuolization. In this same case the left auricle contained a large necrotic area involving the whole thickness of the wall; there was hæmorrhage into all the necrotic areas; the cocci were found throughout the myocardium, mainly in the leucocytes. Iwanoff [57] reports finding a small abscess in the wall of the left ventricle. Winterberg [108] also describes numerous purulent foci in the myocardium. Derignac [28] and many others report hypertrophy of the ventricles.

The pericardium shows changes in about 40 per cent. of the cases. Ecchymoses in both visceral and parietal layers have been observed; our case had a few on the parietal layer; in Frendel's [39] case they were diffuse. Exudative pericarditis is the most common affection; it may be serous (Prochaska,[81] Boyd,[14] Rendu and Halle [83]); hæmorrhagic (Harris and Dabney [46]); purulent (de Beurmann [16]), or fibrinous (Krause,[62] Councilman [26]). Combinations of these may occur, as in the cases of Councilman and of Thayer and Lazear.[97] In de Beurmann's case, gonococci were found in the exudate.

Skin.—The skin eruptions are not constant, but are frequent enough to permit of mention here. Buschke [17] groups all the skin lesions of gonorrhœa as follows:

(1) Simple erythemata.
(2) Those similar to urticaria and erythema nodosum.
(3) Hæmorrhagic and bullose exanthemata.
(4) Hyperkeratoses.

The erythemata and exanthemata are most frequently found in cases of general infection. Erythema scarlatiniforme is occasionally found.[56] Stein [94] reports a case in which there were petechiæ and macules scattered all over the body; in our case there appeared hæmorrhagic vesicles over various areas, especially about the joints. In Abrams's [1] case there were petechiæ all over the body, resembling those seen in measles, which later became pustular and resembled variola; Paulsen's [78] case (probably one of pyæmia) had papules and vesicles on the legs and face; Heller [50] reports an indefinite exanthema on the hands and feet. Dufour [32] describes urticaria occurring during general infection. Schaltz [90] reports erythema nodosum, with recognition of the cocci in the blood. Boettcher [13] cites a case having an eruption on the plantar and palmar surfaces; at first it consisted of efflorescent spots, which later became yellowish and pustular. Finger [36] describes phlegmonous conditions over the joints in a case of mixed infection with the gonococcus and streptococcus.

It seems likely that these skin lesions are comparable in origin to the "rose spots" of typhoid fever, and that they may go on to suppuration either independently or by the aid of other pyogenic cocci.

Liver.—The liver is usually the seat of a not very severe form of cloudy swelling and a fairly constant fatty change. In the majority of the autopsy records no mention is made of this organ. Thayer [98] records passive congestion and possibly a fatty metamorphosis. His [49] reports fatty degeneration; Stein,[94] parenchymatous degenerations; Harris and Dabney,[46] chronic passive congestion and fatty change; Berg,[9] large, fatty liver; Frendel,[39] cloudy swelling; Krause,[62] fatty liver; our case,[100] showed slight central necrosis, and infiltration with polymorphonuclear leucocytes in the Glissonian tissue at the periphery of the lobules.

The fatty change may be consequent upon passive congestion, or both this and the parenchymatous degeneration may be the results of toxæmia; in some forms of which, according to Wells,[110] the

toxins, by acting upon the oxidizing enzymes, may inhibit oxidization and synthetic metabolism; while permitting the cell lipase to form fat and the cell proteases to destroy protoplasm.

Spleen.—The most common findings in this organ are acute tumor and infarction. Of twenty-five cases in which the spleen is accurately described, acute swelling is present in all and infarction in twenty. The infarcts may be hæmorrhagic (Bjelogoloswy [12]), but they are generally anæmic; they may be multiple or single, and seem not to undergo suppuration. Histologically they do not differ from other infarcts of this organ and are undoubtedly due to embolism by fragments from the necrotic heart vegetations. The swelling in those cases closely examined is seen to be due mainly to congestion (Berg, Lartigau, Hunter [55]).

In our case there were also many polymorphonuclear leucocytes and large phagocytes (endothelial cells?) in the splenic pulp; both kinds of cells contained diplococci in great numbers. Seigheim [92] also recovered gonococci from the spleen. Von Leyden [73] reports the finding of many hæmatoidin crystals throughout the pulp.

Kidneys.—The peculiar conditions found in the kidneys of our case led to particular investigation of the literature upon this subject. In this case the kidneys were described at autopsy as greatly enlarged (600 grams) and firm; the cut surface was yellow and mottled with red and pink; blood vessels congested; glomeruli glistening, some red, some pale; the capsule stripped easily. Both answered the same description.

Microscopically, they showed a marked congestion, with a large proportion of leucocytes in the blood. The convoluted tubules were filled with leucocytes, 90 per cent. of which were polymorphonuclears; the remainder were lymphocytes and plasma cells. There were also numbers of these same cells in the glomeruli, with but a very few red blood corpuscles. In many of the Malpighian corpuscles there was a proliferation of the cells of Bowman's capsule, forming the so-called "crescents." There was very little degeneration in the tubular epithelium, and practically no interstitial change. The straight tubules were normal, and contained no abnormal substances. Sections stained with sudan III showed a few fat droplets in the cells of some convoluted tubules. The blood vessels were normal. The presence of so many leucocytes in the convoluted

tubules and the absence of parenchymatous degeneration to any marked degree and the proliferation of the capsules suggest the following statements:

(1) The cocci or their toxins are excreted by the kidney.

(2) The toxic agent is either eliminated through the glomeruli, where it causes a change, or it passes through the walls of the convoluted tubules without exerting a poisonous action on this variety of epithelium. The toxæmia caused by gonococci is mild, as is evidenced by the chronic course of most cases; this mildness may in part be due to the ingestion of the organisms by the phagocytes, hence the toxin may be of such a strength and character as to cause proliferation of the capsular epithelium without causing degeneration of any kidney structure.

The above findings have not been exactly the same as those reported by other observers, as a few cases will illustrate. Fenwick and Cook[35] found large, inflamed kidneys; Colombini,[22] albuminous and cellular casts; Stein,[94] parenchymatous degeneration; von Leyden,[73] hyperæmic, swollen kidneys, with small foci of inflammation (probably very similar to our case); His,[49] subacute parenchymatous nephritis; White,[109] kidneys large and pale, with small subcapsular hæmorrhages and degeneration of the epithelium; Loeb,[70] parenchymatous nephritis; Boyd,[14] acute nephritis and cloudy swelling; Frendel,[39] cloudy swelling. Harris and Dabney (*loc. cit.*) report a case very similar to ours, but it is probably due to a mixed infection.

In many of these cases the septicæmia was not uncomplicated, and when one considers how often the kidneys are the seat of changes whose etiology is obscure, it is usually difficult to say just what conditions are referable to any infectious disease. In our case, however, the infection was single, and the kidneys showed no chronic alterations; hence it seems likely that they show the true picture of how kidneys are affected in uncomplicated gonococcal septicæmia. Also, it may be added, the peculiarities of glomerulonephritis do not appear prominently on gross inspection; therefore, probably a careful examination of many of the above mentioned cases might have revealed conditions similar to those observed by us.

Adrenals.—Only one adrenal was examined in our case. It was firm and redder than normal; microscopically, there appeared a

great congestion and even more marked leucocytosis than was noted in the kidney; there were small extravascular collections of leucocytes in the medulla. Granular parietal thrombi occupied the large vessels of the central portion of the organ, leaving only very small lumina which were filled with fibrinous clots. No similar condition of this viscus has been reported heretofore.

Joints.—The pathology of arthritis preceding or accompanying septicæmia does not differ from that occurring independently of general infection. In the case of Councilman (*loc. cit.*), 100 cc. of gelatinous pus were obtained from the knee joint, and there was purulent infiltration of the muscles above the joint. Bujvid [15] also reports a case in which there were peri-articular abscesses. Arthritis is by no means a constant precursor or even concomitant of the septic condition (Ghon and Schlagenhaufer [42]); nor is it necessarily exacerbated by the onset of general symptoms (Bjelogolowsy [12]). We may conclude with Jadasshon [58] that arthritis is a pyæmic and not a septicæmic condition.

Brain.—Embolism and resulting paralysis have been reported by Heller [50] and Cornell.[25] Probably, also, the case of Cordell [24] in which there was a paralysis of the face muscles, comes under this classification.

Summary.—We may conclude, therefore, that general infection with the gonococcus gives rise to an intoxication, which, in its results, is similar to septicæmias from other pyogenic organisms; but probably, when closely studied, has enough individuality to distinguish it from them. We believe that the glomerulo-nephritis, accompanied by leucocytes in the tubules, will be found to be frequent and characteristic. Retrogressive changes in the liver, and splenic tumor, seem also to be constant findings. In endocarditis caused by other acute infections the mitral valve is most often affected; the cases so far reported show that in gonococcal endocarditis the aortic valves are more frequently attacked than the mitral. The conditions described by us in the adrenal have not been observed by others, hence no statement as to their significance can be advanced.

These changes, with the identification of the organism in the blood stream or in the heart lesion, give a rather distinct picture of the anatomy of gonococcus septicæmia.

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BIBLIOGRAPHY.

[1] Abrams. New York Med. Journ., Aug. 29, 1896.
[2] Ahman. Arch. für Derm. und Syph., Bd. 39, 1897.
[3] Almkvist. Arch. für Derm. und Syph., Bd. 49, 1899.
[4] Anderson. Canada Lancet, Nov. 1896.
[5] Brehmer. Cent. med. Woch., No. 2, 1905.
[6] Bailey. Cincinnati Lancet-Clinic, N. S. 44, 1900.
[7] Balzer. Wien. med. Presse, No. 43, 1900.
[8] Becker. Ber. klin. Woch., 1905, p. 521.
[9] Berg. Med. Record, 1899.
[10] Berg. Wien. med. Woch., liv, p. 65, 1904.
[11] Bertrand. Centralblatt für die Harn und Sex, Org. xii, 1901.
[12] Bjelogolowsy. Deut. Medizinalzeit, No. 102, 1899.
[13] Boettcher. St. Petersburg med. Woch., p. 69, 1899.
[14] Boyd. Boston Med. and Surg. Journ., p. 214, 1896.
[15] Bujvid. Centralblatt für Bakt., p. 435, 1895.
[16] de Beurmann. Berl. klin. Woch., p. 431, 1898.
[17] Buschke. Arch. für Derm. und Syph., Bd. 48, 1899.
[18] Cardile. American Year-Book, 1901.
[19] Cecil. Louisville Jour. Med. and Surg., vol. xi, p. 245.
[20] Crosby. Am. Jour, Med. Sc., cxxix, p. 880, 1905.
[21] Classen. Albany Med. Annals, p. 51, 1890.
[22] Colombini. Centralblatt für Bakt., Bd. xxiv, p. 955.
[23] Combemale. Bull. méd. du Nord., June, 1896.
[24] Cordell. Maryland Med. Jour., p. 121, vol. xxv.
[25] Cornell. Montreal Med. Jour., p. 100, 1900.
[26] Councilman. Am. Jour. Med. Sc., vol. cvi, p. 277.
[27] Dauber und Borst. Deut. Arch. für klin. Med., No. 56.
[28] Derignac. Gaz. méd de Paris, No. 7, 1884.
[29] Doleris. Présse Médicale, July, 1900.
[30] Donnell. Med. Record, Oct., 1890.
[31] Dowd. Jour. Cutan. and Janito-Urinary Diseases, p. 26, 1900.
[32] Dufour. Bull. Soc. de la Rochelle, 1900.
[33] De Fleury. Jour. méd. de Bordeaux, p. 65, 1883–4.
[34] Eulenberg. Deut. med. Woch., p. 919, 1900.
[35] Fenwick and Cook. Brit. med. Jour., Dec., 1899.
[36] Finger. Arch. für Derm. und Syph., xxxviii, 1895.
[37] Fowler. Med. Times, p. 333, xxxiii.
[38] Fürth and Weber. Edinburg. Med Jour., N. S. xviii, p. 33.
[39] Frendel. Wien klin. Woch., No. 27, 1903.
[40] Good. Med. Herald, St. Joseph, No. 24, 1905.
[41] Inaug. Dissertation, Berlin, 1893.
[42] Ghon von Schlagenhaufer. Wien klin. Woch., No. 16, 1898.
[43] Hamilton. Brit. Med. Jour., April, 1905.
[44] Hansteen. Arch. für Derm. und Syph., No. 88, 1897.

[45] Harbitz. Deut. med. Woch., p. 121, 1899.
[46] Harris and Dabney. Johns Hopkins Hosp. Bull., p. 68, 1901.
[47] Hektoen. Cincinnati Lancet-Clinic, p. 451, 1893.
[48] Hewes. Bost. Med. and Surg. Jour., 1894.
[49] His. Berlin klin. Woch., No. 40, 1892.
[50] Heller. Berlin klin. Woch., No. 5, 1898.
[51] Höck. Wien. klin. Woch., No. 41, 1893.
[52] Honl. Centralbl. für Bakt., p. 305, 1899.
[53] von Hoffmann. Centralbl. für die Grenzebiete Med u. Chir., 1903.
[54] Huber. Arch. für Kinderheilkunde, p. 919, 1904.
[55] Hunter. Brit. med. Jour., p. 525, 1905.
[56] Hyde and Montgomery. Disease of the Skin, 1904.
[57] Iwanoff. Wojenno Med. Jour., No. 6: 1898.
[58] Jadasshon. Arch. für Derm. und Syph., No. 43, 1898.
[59] Jicinski. Jour. Amer. Med. Assoc., vol. xxxii, 1899.
[60] Karageosjanz. Eshenedelnik, No. 46, 1899.
[61] Keller. Deut. Arch. für klin. Med., Bd. lvii, 1896.
[62] Krause. Berl. klin. Woch., No. 19, 1904.
[63] Kuchoveski. Progrés médicale, p. 52, 1900.
[64] Lang. Arch. für Derm. und Syph., Bd. xxv, 1893.
[65] Lartigau. Am. Jour. Med. Sc., p. 521, cxxi.
[66] Lenhartz. Berl. klin. Woch., p. 1138, 1897.
[67] Lewis. Lewis. Jour. Cutan. and Genito-Urinary Diseases, p. 298, 1900.
[68] Lichtenstern. Prag-med. Woch., No. 43, 1898.
[69] Loewenhardt. Wien. med. Presse, No. 6, 1898.
[70] Loeb. Deut. Arch. fürr klin. Med., Bd. lxv, 1900.
[71] Loxton. Brit. Med. Jour. vol. i, 1895,
[72] Lody. Medical Age, Detroit, p. 447, xxii.
[73] von Leyden. Deut. med. Woch., p. 909, 1893.
[74] McCaskey. Medical Record, 1900.
[75] McDonnell. Am. Jour. Med. Sc., p. 469, 1900.
[76] Michaelis. Zeitschrift für klin. Med., p. 556, 1896.
[77] Neisser. Berl. klin. Woch., March, 1901.
[78] Paulsen. Münch. med. Woch., No. 47, 1900.
[79] Perin. Ann. de Derm. et Syph., 1890.
[80] Prochaska. Arch für klin. Med., Leipzig, 1905.
[81] Prochaska. Arch für Path. Anat., p. 492, No. 164,
[82] Rana. Arch. für Derm. und Syph., Bd. 39, 1897.
[83] Rendu et Halle. Bull. des Hôp. de Paris, 1897.
[84] Robinson. Med. News, 1896.
[85] Rogers and Torrey. Jour. Amer. Med. Assoc., Jan., 1906.
[86] Rosenthal. Monatsch. für Prakt., Derm., 1900.
[87] Reye. Münch. med. Woch., p. 2202, 1903.
[88] Rosenthal. Berl. klin. Woch., 1900.
[89] Schäffer. Lubarsch-Ostertag's Ergebnisse, p. 617, 1900–01.
[90] Schaltz. Zeitschrift für Prakt., Aertze, 1899.
[91] Sears. Report Boston. City Hosp., vol. ix, 1898.
[92] Seigheim. Berl. klin. Woch., April. 1897.
[93] Seigheim. Zeitschr. für klin. Med., No. 34, 1898.

[94] Stein. Wein klin. Woch., 1900.
[95] Sturgis. Am. Jour. Urology, p. 349, 1904–5.
[96] Thayer and Blumer. Arch. de Méd. Expér., vii, p. 701, 1895.
[97] Thayer and Lazear. Jour. Exp. Med., vol. iv, 1899.
[98] Thayer. Am. Jour. Med. Sc., p. 751, cxxx, 1905.
[99] Tenney. Boston. Med. and Surg. Jour., 1902.
[100] Tyree. Trans. Chicago Path. Soc., Dec. 11, 1905.
[101] Unger. Berl. klin. Woch., 1901.
[102] Ullman. Deut. Arch. für klin. Med., 1901.
[103] Vidal. Ann. de Derm. et Syph., 1893.
[104] v. der Velden. Münch. med. Woch., 1887.
[105] Waite. Jour. Kansas Med. Soc., p. 426, 1905.
[106] Wertheim. Arch. f. Derm. und Syph., 1899, No. 51.
[107] Wassermann. Münch. med. Woch., No. 8, 1901.
[108] Winterberg. Festschrift des Vereins der Aerzte in San Francisco, 1884.
[109] White. Lancet, 1896.
[110] Wells. Jour. Amer. Med. Assoc., Feb. 3, 1906.
[111] Wynn. Lancet, p. 352, vol. i, 1905.
[112] Young. Festschrift for W. H. Welch, 1900.
[113] Zawadsky und Bergen. Wien med. Woch., Nos. 8–9, 1896.

THE BONE-MARROW

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Hæmatology, the study of the blood in health and in disease, is now one of the most important of the recognized branches of medical science, both as a help to the study and diagnosis, and as a guide to the treatment, of disease. Changes may occur in the blood, of such a profound nature and with such rapidity, that it is of fundamental importance to direct our attention not only to the condition of the blood itself, but also to the mechanism by which these changes are brought about and to the study of the tissues of the body in which they occur. Hence the investigation of the physiological and pathological variations in structure and reaction of the bone-marrow, where the great majority of the formed blood-elements is produced, is well worthy of our attention, more especially as it is perhaps, with the exception of the blood itself, the most rapidly changeable and unstable tissue in the body.

A thorough examination of the bone-marrow should embrace the description of the naked-eye and microscopic appearances both in film preparations and in sections, together with the study of the physical and chemical changes which occur in the tissue. My attention, however, has been more especially occupied by the first three of these subdivisions of the subject.

It is convenient to describe first the varieties of *bone-marrow;* then to enumerate the different varieties of cell which may be found in the tissue; next we shall deal with the most important reactions and degenerations to which it is liable under pathological conditions; after which I shall briefly recapitulate the more important of the known *functions* of the tissue.

VARIETIES OF BONE-MARROW

(1) Primitive or Embryonic Marrow.—The medullary cavities of the bones are, early in development, filled with a tissue com-

posed of cells, mucoid or myxomatous in type, possessing numerous branching and interlacing processes. These are in histological structure and appearance similar to the cells found in other mucoid tissues in the fœtus, and in this situation they afterwards develop into the adenoid reticulum or connective tissue framework of the marrow, in the interstices of which appear, about the fourth month of fœtal life or a little earlier, the hæmopoietic cells proper. By the proliferation of these cells is formed the next variety of marrow.

(2) Red, "Lymphoid," or Formative Bone-marrow proper, in which, except during fœtal and perhaps early infant life when the liver and spleen still retain some of their primitive hæmopoietic functions, are developed all the red blood cells; the majority, if not all of the granular leucocytes; and possibly also some of the non-granular white cells of the blood.

In the infant and young child this type of marrow is found throughout the interior of the entire osseous system, but, as age advances, in certain situations, especially within the central parts of the shaft of the long bones, the red marrow tends to undergo a process of physiological "degradation," the hæmopoietic cells almost entirely disappearing from large tracts of the tissue, and the space being filled up by a development of fat within the connective tissue cells of the adenoid reticulum. Usually, however, *some* of the blood-forming cells remain towards the periphery of the shaft, and also more especially towards the ends of the long bones, a point of extreme importance as will be seen later, as it is from these positions that proliferation may again take place should there be any special demand for increased production of blood cells. In the short, flat, and irregular bones, sternum, ribs, vertebra, etc., the red marrow undergoes a much less degree of this fatty change, though even in these situations there is a progressive increase in its amount, which may become very considerable in advanced age and in some pathological conditions. There is therefore in the red as well as in the fatty marrow a potentiality for the proliferation of the blood-forming elements, and for a corresponding diminution or absorption of the fat cells, should there be a necessity for increased output of the blood cells found in the marrow.

(3) Yellow or Fatty Marrow.—Found, as age advances,

more especially in the central parts of the shafts of the long bones, as above described, the fat being developed within the cells of the adenoid reticulum. These areas of fatty marrow form a most important "potential area" in which the development of blood-forming cells can be carried on should there be any special call for these cells to combat the attack of disease, or to replace cells destroyed by its action, for example in many of the acute and chronic diseases characterized by leucocytosis, or by anæmia. Any factors interfering with this reaction, *e.g.*, chronic degenerative conditions of the marrow such as may be found in syphilis, chronic alcoholism, lead poisoning, malignant disease, etc., may lead to a rapidly fatal issue if such a patient contracts an acute febrile disease such as pneumonia. Two of the most important of such degenerative conditions of the marrow may next be described.

(4) Fibroid Marrow.—In old persons, or earlier in life under certain pathological conditions, and more especially in syphilis, just as in other highly organized tissue of the body, the connective tissue elements may be found proliferating at the expense of the more highly endowed and therefore more delicate and more easily destroyed functioning cells of the part. This change may be due to the direct action of some toxic substances, as well as to diminished nutrition from vascular and other derangements.

(5) Gelatinous Marrow.—An important degenerative change, which may supervene in old age, and especially in cases of chronic debilitating disease accompanied by anæmia, such as cancer, phthisis, etc., or in starvation. It may also occur very rapidly in acute disease such as ulcerative endocarditis, pneumonia, puerperal and other forms of septicæmia, diphtheria, etc., where the organisms or their toxins possess a high degree of virulence.

Gelatinous degeneration may attack either yellow or red marrow, and seems specially liable to be found when the former has previously undergone prolonged leucoblastic transformation, where the proliferative changes have not been kept up, *i.e.* an exhaustion supervening in an over-active marrow in which the strain cannot be maintained. It is important also to bear in mind that the condition may be brought about artificially by certain drugs which at first, when given in small therapeutic doses, may stimulate the hæmopoietic functions of the marrow, *e.g.*, arsenic, lead, mer-

cury, etc., but the prolonged application or excessive doses of which may bring about profound gelatinous degeneration, accompanied by complete disappearance of the blood-forming cells.

VARIETIES OF CELL FOUND IN THE MARROW.

Only a bare enumeration of the cells found in the marrow is possible in a short résumé of the subject such as this. In film preparations and in sections of red or blood-forming bone-marrow, the following varieties of cells may be distinguished:

(I) Blood-forming Cells:

(A) *Leucocyte Series:*

(a) Non-granular cells with basophilic protoplasm:

(1) Large.

(2) Small:

(i) Cells similar to, but smaller than, the large variety.

(ii) Cells identical in appearance and in staining reaction with the small lymphocyte of the blood.

(b) Granular Cells:

(1) Neutrophile (corresponding to Amphophile in the rabbit):

(i) Myelocytes, with large, rounded or oval nucleus.

(a) Larger variety.

(b) Smaller variety.

(ii) Intermediate Cells, with indented or horseshoe-shaped nucleus.

(iii) Polymorphonuclear Cells, or fully developed Leucocytes.

(2) Eosinophile:

(i) Myelocytes.

(ii) Intermediate Cells.

(iii) Polymorphonuclear Cells or Leucocytes.

(3) Basophile:

(a) Mastcells.

(i) Myelocytes.

(iii) Polymorphonuclear Cells.

(b) Cells resembling the eosinophil myelocytes, but with granulations staining with the basic dye.

(B) *Hemoglobin-holding Series*:

(1) Normoblasts, developing into normocytes or ordinary red blood corpuscles.

(2) Megaloblasts, normally giving rise to normoblasts, or, under certain pathological circumstances, *e.g.,* in pernicious anæmia, producing Megalocytes.

(3) Gigantoblasts, developing into Gigantocytes—these being merely very large forms of the preceding variety of cell, found especially in Pernicious Anæmia.

(II) Giant Cells:

(1) Mononucleated.—"Megakaryocytes," with single, highly complex, basket-like nucleus.

(2) Multinucleated.—"Polykaryocytes," *e.g.,* osteoclasts, etc.

(III) Cells of Connective Tissue Type:

(1) Fat Cells.

(2) Cells of the Adenoid Reticulum.

(3) Various forms of Phagocytic Cell (pigmented cells, etc.).

(4) Ordinary Connective Tissue Cells passing in with the blood-vessels, etc.

(IV) Endothelial Cells, lining the walls of capillaries, blood sinuses, etc.:

(1) Found in their normal positions in the vessel walls.

(2) Found proliferating, taking on phagocytic functions, etc.

REACTIONS OF THE BONE-MARROW IN DISEASES

In many diseased conditions, rapid proliferative changes may occur both in the red and in the yellow marrow, and according as the change is characterized by the increased production of *red* or of *white* blood cells, the process may be described as being either *erythroblastic* or *leucoblastic* in type. In health there is in the marrow a normal balance between these two processes, and usually, when the tissue is first stimulated to proliferate, there is increased production of *all* varieties of marrow-formed blood cells. Soon, however, in most cases, the tissue is found to produce especially the variety of cell for which there is special demand, and the reaction becomes either leuco- or erythro-blastic in type, though in some cases *both* changes may occur simultaneously in the same marrow. A very common sequence of events which may be found in certain chronic septicæmias and other diseases, is, with the occurrence of a polymorphonuclear leucocytosis, a slight transitory increase in the number of red cells in the peripheral blood. Then, if the leucocytosis

has to be kept up for a prolonged period, the reds become progressively diminished in numbers. If the marrow be examined, corresponding phenomena will be found occurring, *i.e.,* the leucoblastic will predominate over and obscure the erythroblastic type of reaction and, if the leucocytosis has to be kept up for a still longer period, degenerative changes may occur in the leucoblastic marrow, and a progressive anæmia, characterized by transformation to the erythroblastic type of change, may supervene. Then, as the anæmia becomes more profound, this too may pass off as the marrow becomes exhausted, and the blood-forming tissue may undergo gelatinous degeneration.

Leucoblastic Marrows.—Corresponding roughly to the different types of leucocytosis observed clinically, we may subdivide the leucoblastic changes found in the marrow into four groups:

(1) *Neutrophile leucoblastic reaction.*—The type found more especially in connection with the establishment of an *inflammatory leucocytosis, i.e.,* an increased production of the neutrophile series of cells, a condition found in the majority of acute infective diseases, whether these be local (*e.g.,* inflammation, abscess formation, etc., due to the presence of staphylo-and strepto-cocci, B. coli, and many other microörganisms): or general conditions, such as pneumonia, the septicæmias, etc., the results produced in the marrow being practically identical in both sets of cases, differing rather because of the intensity, quality and amount of the irritant, than on account of its special point of attack and distribution in the body. This leucoblastic reaction is, of course, naturally most readily produced in the red marrow, and shows as an increased proliferation of the special variety of hæmopoietic cell required—in this case the neutrophilic myelocytes. But if further demands are made upon the tissue, the change is then found to spread into the fatty or yellow marrow in the shafts of the long bones and elsewhere, and in these situations all degrees of the reaction may be observed, up to complete transformation to fully formed red marrow. This transformation is found to spread into the fatty marrow from the periphery and ends of the shaft in the case of the long bones, *i.e.,* from the situations in which the hæmopoietic cells had not completely disappeared, when the physiological degradation of the marrow occurred with advancing age. Any pathological conditions preventing such a

leucoblastic reaction on the part of the marrow, *e.g.*, the presence of fibroid or gelatinous degeneration, may determine a rapidly fatal issue in a case, say, of pneumonia, diphtheria, etc., characterized clinically by the absence of leucocytosis (*aleucocytosis*), or even by leucopœnia or an actual diminution of the leucocyte count.

(2) *Eosinophile leucoblastic reaction.*—Characterized by special increase of the eosinophile granular cells.

(3) *Blasophile leucoblastic reaction.*—Characterized by special proliferation of the basophile granular cells, a condition found particularly in myelogenous leukæmia, and in certain nutritional disorders.

(4) *Hyaline leucoblastic reaction.*—Characterized by an increase in the numbers of the pre-myelocytes or myeloblasts, *i.e.*, large basophile non-granular cells from which the cells of the granular series are developed. This type of reaction is specially found in marrows from cases of prolonged leucocytosis, *e.g.*, in chronic septicæmias or pyemias, malignant disease, etc., and in its most extreme form is observed in cases of so-called acute "lymphatic" leukæmia.

Erythroblastic or red blood cell-forming marrows, may be subdivided into two main types:

(1) *Normoblastic.*—*i.e.*, marrows in which normoblasts or normal nucleated red cells, are found in excess, *e.g.*, after hæmorrhage, and in most secondary anæmias, where the tissue endeavors to compensate for the deficiency in the number of the red cells of the blood. We have already noted the fact that this change may also supervene in leucoblastic marrows which have become exhausted.

(2) *Megaloblastic.*—Where a more primitive type of red cell formation is developed. Megaloblasts, or nucleated red cells of abnormally large size, and possessing nuclei of more primitive type, are found increased in a very large number of diseases, both acute and chronic. They are usually present only in small numbers although in some cases of exophthalmic goître, malignant disease, tubercular disease, many acute and chronic septic conditions, typhoid fever, etc., the writer has observed them with comparative frequency, and sometimes in very large numbers. The most extreme degree of megaloblastic transformation, however, is seen in cases

of pernicious anæmia, and in certain secondary anemias closely allied to it, notably in those due to the presence of bothriocephalus latus, ankylostoma duodenale and other intestinal parasites, and also in myelogenous leukæmia.

FUNCTIONS OF THE BONE-MARROW.

These may be briefly summarized as follows:

(1) HÆMOPOIETIC FUNCTION:

(a) *Erythroblastic.*—The formation of red blood cells.

(b) *Leucoblastic.*—The formation of white blood cells of the granular series.

A normal balance between these two processes is maintained in health; whilst in disease there is a latent capacity for the increased production of any or all of the blood cells to meet the increased demand for these, whether this be due to the necessity of replacing cells destroyed by the disease, as in the anæmias, or to the fact that more blood cells are required to combat the attack of the invading bacterial or other pathogenic agent, as is seen in the various inflammatory and suppurative conditions, the fatty marrow forming an important potential area for the production of this reaction in the marrow.

(2) BLOOD DESTRUCTION, PHAGOCYTOSIS, ETC.:

The marrow, like the liver and spleen, constitutes a very important hæmolytic area, and this function is found greatly increased in most of the acute and chronic toxic diseases, *e.g.*, the acute infective fevers, septicemias, etc.; the anæmias, especially pernicious; the cachexias, especially malignant disease, malaria, etc.

Phagocytosis of pathogenic organisms also occurs to a considerable degree in many diseases, the marrow appearing to be specially liable in the presence of microörganisms, and pure cultures of these may frequently be obtained from the tissue, *e.g.*, in pneumonias, streptococcal and other septicæmias, etc. Some blood and other parasites pass certain stages of their development in the bone-marrow, and endothelial and other forms of phagocytic cells may be found containing englobed parasites or their remains, *e.g.*, in malaria, trypanosomiasis, etc.

(3) FORMATION, NUTRITION, AND ABSORPTION OF BONE.—These functions are of great importance, but for the details of these

processes reference must be made to the special physiological works upon this subject. Under certain pathological conditions very rapid absorption of bone may occur, *e.g.*, in the enlargement of the medullary cavity to allow of greater room for the proliferation and expansion of the contained marrow, a process which may in some cases take place with marvellous rapidity. , Many conditions which have previously been regarded as primary diseases of bone, must now be looked upon as coming under this category, *e.g.*, osteomalacia, "osteoporosis," osteitis deformans, and analogous conditions, whilst a somewhat similar absorption of bone is seen in pernicious anæmia.

(4) Functions in Relation to General Nutrition, and Especially With Regard to Metabolism of Fat.—Fatty or yellow marrow is very richly supplied with blood-vessels, and in fevers, etc., the fat may be very rapidly absorbed from the tissue, which thus acts as a store-house for this substance. It is also quite possible that the marrow, like many of the other glandular organs in the body, may give rise to an internal secretion of importance to general nutrition and metabolism, but, owing to the impossibility of experimentally eliminating the action of the tissue on account of its wide anatomical distribution, it is as yet difficult to prove the truth of this supposition.

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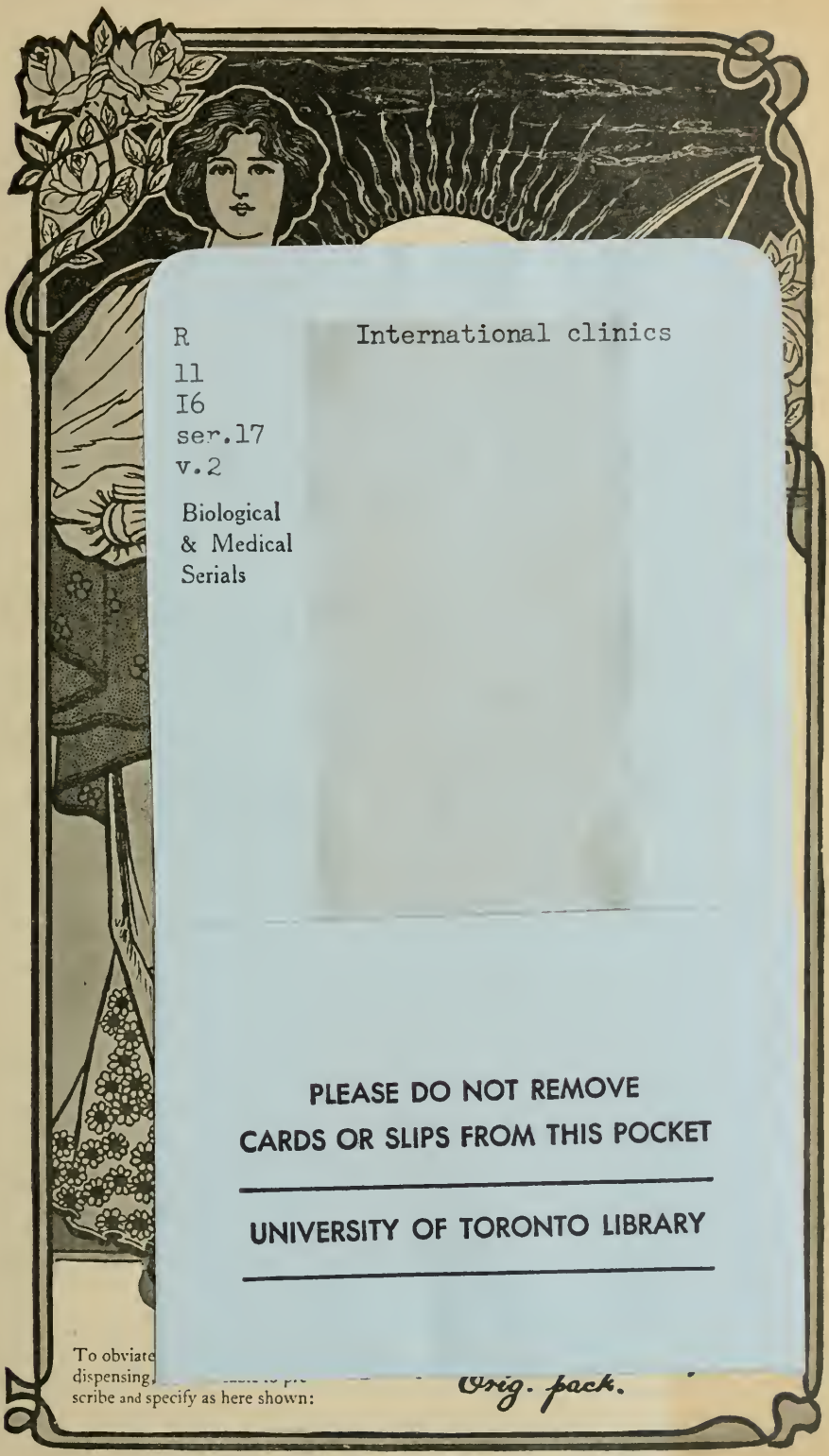

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